

Cardiovascular physiology

- **Left ventricular ejection fraction** = (stroke volume / end diastolic LV volume) * 100%
- **Stroke volume** = end diastolic LV volume - end systolic LV volume

Pulse pressure:

- Pulse pressure = Systolic Pressure - Diastolic Pressure

Factors which increase pulse pressure

- 1) a less compliant aorta (this tends to occur with advancing age)
- 2) increased stroke volume

Hypertention

Secondary causes:

A. Endocrine disorders:

- 1) primary hyperaldosteronism:
 - ☒ It is thought that between 5-10% of patients diagnosed with hypertension have primary hyperaldosteronism, including Conn's syndrome.
 - ☒ This makes it **the single most common cause** of secondary hypertension.
- 2) pheochromocytoma
- 3) Cushing's syndrome
- 4) Liddle's syndrome
- 5) congenital adrenal hyperplasia (11-beta hydroxylase deficiency)
- 6) acromegaly

B. Renal disease:

Accounts for a large percentage of the other cases of secondary hypertension

- 1) glomerulonephritis
- 2) pyelonephritis
- 3) adult polycystic kidney disease
- 4) renal artery stenosis

Other causes include:

- 1) NSAIDs
- 2) steroids
- 3) MAOI
- 4) pregnancy
- 5) the combined oral contraceptive pill
- 6) coarctation of the aorta

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Isolated systolic hypertension(ISH)

- common in the elderly,
- Affecting around 50% of people older than 70 years old.
- The Systolic Hypertension in the Elderly Program (SHEP) back in 1991 established that treating ISH reduced both **strokes** and **IHD**.
- Drugs such as **thiazides** were recommended as first line agents.
- This approach is contradicted by the 2011 NICE guidelines which recommends treating ISH in the same **stepwise fashion as standard hypertension**.

Hypertension diagnosis:

- NICE published updated guidelines for the management of hypertension in 2011.
- Some of the key changes include:
 - ✓ classifying hypertension into stages
 - ✓ recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)

Why were these guidelines needed?

It has long been recognised by doctors that there is a subgroup of patients whose blood pressure climbs 20 mmHg whenever they enter a clinical setting, so called '**white coat hypertension**'. If we just rely on clinic readings then such patients may be diagnosed as having hypertension when the vast majority of time there blood pressure is normal.

This has led to the use of both ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM) to confirm the diagnosis of hypertension. These techniques allow a more accurate assessment of a patients' overall blood pressure. Not only does this help prevent overdiagnosis of hypertension - ABPM has been shown to be a more accurate predictor of cardiovascular events than clinic readings.

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocate.

Stage	Criteria
Stage 1 hypertension	Clinic BP $\geq$ 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP $\geq$ 135/85 mmHg
Stage 2 hypertension	Clinic BP $\geq$ 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP $\geq$ 150/95 mmHg
Severe hypertension	Clinic systolic BP $\geq$ 180 mmHg, or clinic diastolic BP $\geq$ 110 mmHg

Diagnosing hypertension:

- Firstly, NICE recommend measuring blood pressure in both arms when considering a diagnosis of hypertension.
- If the difference in readings between arms is more than 20 mmHg then the measurements should be repeated.
- If the difference remains > 20 mmHg then subsequent blood pressures should be recorded from the arm with **the higher reading**.
- It should of course be remember that there are pathological causes of unequal blood pressure readings from the arms, such as **supraaortic stenosis**.
- It is therefore prudent to listen to the heart sounds if a difference exists and further investigation if a very large difference is noted.
- NICE also recommend taking a **second reading** during the consultation, if the first reading is > 140/90 mmHg. The lower reading of the two should determine further management.
- NICE suggest offering ABPM or HBPM to any patient with a blood pressure $\geq 140/90$
- **If however the blood pressure is $\geq 180/110$ mmHg:**
 - **immediate treatment** should be considered
 - if there are signs of papilloedema or retinal haemorrhages NICE recommend **same day assessment by a specialist**
 - NICE also recommend referral if a **phaeochromocytoma** is suspected (labile or postural hypotension, headache, palpitations, pallor and diaphoresis)

Ambulatory blood pressure monitoring (ABPM):

- at least 2 measurements per hour during the person's usual waking hours (for example, between 08:00 and 22:00)
- use the average value of at least 14 measurements
- If ABPM is not tolerated or declined HBPM should be offered.

Home blood pressure monitoring (HBPM):

- for each BP recording, two consecutive measurements need to be taken, at least 1 minute apart and with the person seated
- BP should be recorded twice daily, ideally in the morning and evening
- BP should be recorded for at least 4 days, ideally for 7 days
- discard the measurements taken on the first day and use the average value of all the remaining measurements

Interpreting the results

1) ABPM/HBPM $\geq 135/85$ mmHg (i.e. stage 1 hypertension)

- **treat if < 80 years of age AND any of the following apply;**
 - ⇒ target organ damage,
 - ⇒ established cardiovascular disease,
 - ⇒ a 10-year cardiovascular risk equivalent to 20% or greater,
 - ⇒ renal disease or diabetes

2) ABPM/HBPM $\geq 150/95$ mmHg (i.e. stage 2 hypertension)

- **offer drug treatment regardless of age**

Hypertension management:

- NICE published updated guidelines for the management of hypertension in 2011.
- **Some of the key changes include:**
 - ✓ classifying hypertension into stages
 - ✓ recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)
 - ✓ calcium channel blockers are now considered superior to thiazides
 - ✓ bendroflumethiazide is no longer the thiazide of choice

Managing hypertension

A. **Lifestyle advice** should not be forgotten and is frequently tested in exams:

- 1) A low salt diet is recommended, aiming for **less than 6g/day**, ideally 3g/day. (The average adult in the UK consumes around 8-12g/day of salt)
- 2) A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, **reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg**
- 3) caffeine intake should be reduced
- 4) the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

B. **ABPM/HBPM $\geq$ 135/85 mmHg (i.e. stage 1 hypertension)**

- **treat if < 80 years of age AND any of the following apply**; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 20% or greater

C. **ABPM/HBPM $\geq$ 150/95 mmHg (i.e. stage 2 hypertension)**

- offer drug treatment regardless of age

 For patients < 40 years consider specialist referral to exclude secondary causes.

NICE have published guidelines on Hypertension (CG127) to aid with choice of antihypertensive

	Age <55 and non black	Age >55 or black
1 st line	A	C or D
2 nd line	A + C or D	C or D + A
3 rd line	A, C, D	A, C, D
4 th line	Add in other agents	Add in other agents

- **Black people of African or Caribbean origin** and **older people** (that is, over 55) have **lower renin states** and ACE inhibitors are generally not as effective as antihypertensives.
- **Diabetes** constitutes a compelling indication for **ACE inhibitors** (or ARBs if ACEi are not tolerated).
- Other compelling indications which would potentially override the ACD guidance above, include;
 - 1) first-line therapy with **alpha-blockers** for hypertensive subjects with **BPH** ;
 - 2) **beta-blockers** for hypertensive subjects with **heart failure** or **angina**, etc

Step 1 treatment:

- patients < 55-years-old: ACE inhibitor (A)
- patients > 55-years-old or of Afro-Caribbean origin: calcium channel blocker or diuretics

Step 2 treatment:

- ACE inhibitor + calcium channel blocker or diuretics

Step 3 treatment:

- A + C + D
- NICE now advocate using either:
 - ✓ **chlorthalidone** (12.5-25 mg once daily) or
 - ✓ **indapamide** (1.5 mg modified-release once daily or 2.5 mg once daily) in preference to a conventional thiazide diuretic such as bendroflumethiazide

NICE define a clinic BP $\geq 140/90$ mmHg **after step 3 treatment** with **optimal or best tolerated doses** as **resistant hypertension**. They suggest **step 4 treatment** or **seeking expert advice**

Step 4 treatment:

- 1) **consider further diuretic treatment**
 - if potassium < 4.5 mmol/l add **spironolactone** 25mg od
 - if potassium > 4.5 mmol/l add **higher-dose thiazide-like diuretic** treatment
- 2) If further diuretic therapy is not tolerated, or is contraindicated or ineffective,
 - ⇒ **consider an alpha- or beta-blocker**

Patients who fail to respond to step 4 measures **should be referred to a specialist**. NICE recommend:

If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.

Blood pressure targets

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Centrally acting antihypertensives:

- 1) **methyldopa**: used in the management of hypertension during pregnancy
- 2) **moxonidine**: used in the management of essential hypertension when conventional antihypertensives have failed to control blood pressure
- 3) **clonidine**: the antihypertensive effect is mediated through **stimulating alpha-2** adrenoceptors in the vasomotor centre

New drugs:

Direct renin inhibitors:

- e.g. **Aliskiren** (branded as Rasilez)
- by inhibiting renin blocks the conversion of angiotensinogen to angiotensin I
- No trials have looked at mortality data yet.
- Trials have only investigated fall in blood pressure.
- Initial trials suggest aliskiren reduces blood pressure to a similar extent as angiotensin converting enzyme (ACE) inhibitors or angiotensin-II receptor antagonists
- adverse effects were uncommon in trials although **diarrhoea** was occasionally seen
- only current role would seem to be **in patients who are intolerant of more established antihypertensive drugs**

Malignant hypertension

- severe hypertension (e.g. **>200/130** mmHg)
- occurs in both essential and secondary types
- **fibrinoid necrosis** of blood vessels, leading to:
 - ⇒ retinal haemorrhages, exudates, and
 - ⇒ proteinuria, haematuria due to renal damage (benign nephrosclerosis).
- can lead to cerebral oedema → **encephalopathy**

Features:

- 1) classically: severe headaches, nausea/vomiting, visual disturbance
- 2) however chest pain and dyspnoea common presenting symptoms
- 3) papilloedema
- 4) severe: encephalopathy (e.g. seizures)

Management:

- 1) bed rest
- 2) reduce **diastolic** no lower than **100mmHg within 12-24 hrs**
- 3) most patients: **oral therapy** e.g. **atenolol**
- 4) if **severe/encephalopathic**: IV sodium **nitroprusside/labetolol**

Hypertension in pregnancy

- NICE published guidance in 2010 on the management of hypertension in pregnancy.
- They also made recommendations on reducing the risk of hypertensive disorders developing in the first place.
- **Women who are at high risk of developing pre-eclampsia:**
 - ⇒ Should take **aspirin 75mg od** from **12 weeks** until the birth of the baby.
 - ☒ **High risk groups include:**
 - 1) hypertensive disease during previous pregnancies
 - 2) chronic kidney disease
 - 3) autoimmune disorders such as SLE or antiphospholipid syndrome
 - 4) DM type 1 or 2
- **Remember, in normal pregnancy:**
 - ✓ blood pressure usually **falls** in the first trimester (particularly the diastolic), and continues to fall until 20-24 weeks
 - ✓ after this time the blood pressure usually **increases to pre-pregnancy levels** by term
- **Hypertension in pregnancy is usually defined as:**
 - ✓ systolic > 140 mmHg or diastolic > 90 mmHg
 - ✓ or an increase above booking readings of > 30 mmHg systolic or > 15 mmHg diastolic

After establishing that the patient is hypertensive they should be categorized into one of the following groups:

Pre-existing HTN	Pregnancy-induced HTN (PIH, gestational HTN)	Pre-eclampsia
• A history of hypertension before pregnancy or • an elevated blood pressure > 140/90 mmHg before 20 weeks gestation • No proteinuria, No oedema • Occurs in 3-5% of pregnancies and is more common in older women	• Hypertension occurring in the second half of pregnancy (i.e. after 20 weeks) • No proteinuria, no oedema • Occurs in around 5-7% of pregnancies • Resolves following birth (typically after one month). • Women with PIH are at increased risk of future ⇒ pre-eclampsia or ⇒ HTN later in life	• Pregnancy-induced HTN in association with proteinuria (> 0.3g / 24 hours) • Oedema may occur but is now less commonly used as a criteria • Occurs in around 5% of pregnancies

Pre-eclampsia

- Pre-eclampsia is a condition seen **after 20 weeks** gestation characterised by:
 - ✓ **Pregnancy-induced HTN** in association with
 - ✓ **Proteinuria (> 0.3g / 24 hours)**.
- Oedema used to be third element of the classic triad but is now often not included in the definition as it is not specific
- **Pre-eclampsia is important as it predisposes to the following problems:**
 - 1) **Fetal:**
 - ⇒ prematurity,
 - ⇒ intrauterine growth retardation
 - 2) **Eclampsia**
 - 3) **Hemorrhage:**
 - ⇒ placental abruption,
 - ⇒ intra-abdominal hemorrhage,
 - ⇒ intra-cerebral hemorrhage
 - 4) **cardiac failure**
 - 5) **multi-organ failure**

Risk factors:

- 1) > 40 years old
- 2) nulliparity (or new partner)
- 3) multiple pregnancy
- 4) pregnancy interval of more than 10 years
- 5) BMI > 30 kg/m²
- 6) pre-existing vascular disease such as HTN or renal disease
- 7) diabetes mellitus
- 8) family history of pre-eclampsia
- 9) previous history of pre-eclampsia

Features of severe pre-eclampsia:

- 1) hypertension: typically > **170/110** mmHg and **proteinuria** as above
- 2) proteinuria: dipstick ++/+++
- 3) headache
- 4) visual disturbance
- 5) papilloedema
- 6) RUQ/epigastric pain
- 7) **hyperreflexia**
- 8) platelet count < **100** * 10⁶/l, **abnormal liver enzymes** or **HELLP** syndrome

Management:

Consensus guidelines recommend treating blood pressure > 160/110 mmHg although many clinicians have a lower threshold

- 1) **Oral labetalol** is now **first-line** following the 2010 NICE guidelines.
- 2) **Nifedipine** and **hydralazine** may also be used
- 3) **Delivery** of the baby is the most important and **definitive management** step. The timing depends on the individual clinical scenario

Eclampsia

- Eclampsia may be defined as the development of **seizures** in association **pre-eclampsia**.
- To recap, pre-eclampsia is defined as:
 - 1) condition seen after 20 weeks gestation
 - 2) pregnancy-induced hypertension
 - 3) proteinuria

Management:

A. **Magnesium sulphate:**

- ⇒ Used to both prevent seizures in patients with severe pre-eclampsia and treat seizures once they develop.
- ⇒ Guidelines on its use suggest the following:
 - 1) should be given once a decision to deliver has been made
 - 2) in eclampsia an **IV bolus** of **4g over 5-10 minutes** should be given followed by an **infusion** of **1g / hour**
 - 3) urine output, reflexes, respiratory rate and oxygen saturations should be monitored during treatment
 - 4) treatment should **continue for 24 hours** after last seizure or delivery (around 40% of seizures occur post-partum)

B. Other important aspects of treating severe pre-eclampsia/eclampsia include **fluid restriction** to avoid the potentially serious consequences of fluid overload

Management of hypertension in Diabetes mellitus

NICE recommend the following blood pressure targets for type 2 diabetics:

- if end-organ damage (e.g. renal disease, retinopathy) < **130/80** mmHg
- otherwise < **140/80** mmHg

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- A 2013 Cochrane review casted doubt on the wisdom of lower blood pressure targets for patients with diabetes.
 - It compared;
 - ⇒ patients who had tight blood pressure control (targets < 130/85 mmHg) with
 - ⇒ More relaxed control (< 140-160/90-100 mmHg).
 - Patients who were more tightly controlled had a **slightly reduced rate of stroke** but otherwise outcomes were not significantly different.
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- Because ACE-inhibitors have a **renoprotective effect** in diabetes they are the **first-line** antihypertensives recommended for NICE.
 - Patients of African or Caribbean family origin should be offered an **ACE-inhibitor** plus either **a thiazide diuretic** or **calcium channel blocker**.
 - Further management then reverts to that of non-diabetic patients, as discussed earlier in the module.
 - Remember that autonomic neuropathy may result in more postural symptoms in patients taking antihypertensive therapy.
 - The routine use of **beta-blockers** in uncomplicated hypertension should be avoided, particularly when given in combination with **thiazides**, as they may cause **insulin resistance**, **impair insulin secretion** and **alter the autonomic response to hypoglycaemia**.

Heart Failure

NYHA classification

The New York Heart Association (NYHA) classification is widely used to classify the severity of heart failure:

NYHA Class I:

- **no symptoms**
- **no limitation**: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations

NYHA Class II:

- **mild symptoms**
- **slight limitation** of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations or dyspnoea

NYHA Class III:

- **moderate symptoms**
- **marked limitation** of physical activity: comfortable at rest but less than ordinary activity results in symptoms

NYHA Class IV:

- **severe symptoms**
- unable to carry out any physical activity without discomfort: **symptoms** of heart failure are present even **at rest** with increased discomfort with any physical activity

Diagnosis Heart failure

- NICE issued updated guidelines on diagnosis and management in 2010.
- The choice of investigation is determined by whether the patient has previously had a **myocardial infarction** or not.

1) **Previous myocardial infarction** who have suspected heart failure:

⇒ arrange echocardiogram and specialist review within **2 weeks**

2) **No previous myocardial infarction** who have suspected heart failure:

- measure serum natriuretic peptides (**BNP**)
 - ⇒ if levels are **'high'** arrange echocardiogram within **2 weeks**
 - ⇒ if levels are **raised** arrange echocardiogram within **6 weeks**

- Patients with **normal BNP** concentrations should be investigated for other causes of their symptoms as a normal BNP makes **heart failure unlikely**.
- 40% of patients with raised BNP will turn out to have left ventricular systolic dysfunction (LVSD) on echocardiographic assessment.

Serum natriuretic peptides (BNP)

- B-type natriuretic peptide is a **hormone** Produced mainly by the **left ventricular myocardium** in response to **strain**.
- **Very high levels** are associated with a **poor prognosis**.

	BNP	NTproBNP
High levels	> 400 pg/ml (116 pmol/litre)	> 2000 pg/ml (236 pmol/litre)
Raised levels	100-400 pg/ml (29-116 pmol/litre)	400-2000 pg/ml (47-236 pmol/litre)
Normal levels	< 100 pg/ml (29 pmol/litre)	< 400 pg/ml (47 pmol/litre)

Factors which alter the BNP level:

Increase BNP levels	Decrease BNP levels
1) Left ventricular hypertrophy 2) Right ventricular overload 3) diastolic dysfunction 4) Tachycardia, AF 5) Aortic stenosis, 6) Ischaemia 7) ACS, stable angina, 8) HTN, Diabetes 9) Sepsis 10) Hypoxaemia (including pulmonary embolism) 11) COPD, cor pulmonale 12) Age > 70, women > men 13) GFR < 60 ml/min (Acute & CRF) 14) Liver cirrhosis 15) Hyperaldosteronism, Cushing S	1) Obesity 2) Diuretics 3) ACE inhibitors 4) Angiotensin 2 receptor blockers 5) Beta-blockers 6) Aldosterone antagonists

Heart failure drug management:

- A number of drugs have been shown to improve mortality in patients with chronic heart failure:
 - 1) **ACE inhibitors** (SAVE, SOLVD, CONSENSUS)
 - 2) **beta-blockers** (CIBIS)
 - 3) **spironolactone** (RALES)
 - 4) **hydralazine** with **nitrates** (VHEFT-1)
- **No long-term reduction** in mortality has been demonstrated for **loop diuretics** such as furosemide.

NICE issued updated guidelines on management in 2010, key points include:

- 1) **first-line treatment** for all patients is **both** an **ACE-inhibitor** and a **beta-blocker**
- 2) **second-line treatment** is now either:
 - ⇒ **angiotensin II receptor blocker**
 - ⇒ **an aldosterone antagonist**, or
 - ⇒ **a hydralazine** in combination with a **nitrate**
- 3) **if symptoms persist** cardiac resynchronisation therapy or **digoxin*** should be considered
- 4) **diuretics** should be given for fluid overload
- 5) offer **annual influenza vaccine**
- 6) offer one-off **pneumococcal vaccine**

Adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years

*digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties. Digoxin is strongly indicated if there is coexistent atrial fibrillation

Prescribing in patients with heart failure

The following medications may exacerbate heart failure:

- 1) **Thiazolidinediones**: pioglitazone is contraindicated as it causes fluid retention
 - 2) **Verapamil**: negative inotropic effect
 - 3) **class I antiarrhythmics**; **flecainide** (negative inotropic and proarrhythmic effect)
 - 4) **NSAIDs/glucocorticoids**: should be used with caution as they cause fluid retention
- Low-dose aspirin is an exception - many patients will have coexistent cardiovascular disease and the benefits of taking aspirin easily outweigh the risks

pioglitazone is now the only thiazolidinedione on the market

Angiotensin-converting enzyme inhibitors

- ACE inhibitors are now the established **first-line treatment** in younger patients with **hypertension**
- also extensively used to treat **heart failure**
- They are known to be less effective in treating hypertensive Afro-Caribbean patients.
- ACE inhibitors are also used to treat **diabetic nephropathy** and have a role in secondary prevention of **ischaemic heart disease**.

Mechanism of action:

- inhibit the conversion angiotensin I to angiotensin II

Side-effects:

- 1) **Cough:**
 - ⇒ occurs in around 15% of patients and
 - ⇒ May occur up to a year after starting treatment.
 - ⇒ Thought to be due to increased **bradykinin** levels
- 2) **angioedema:** may occur up to a year after starting treatment
- 3) **hyperkalaemia**
- 4) **first-dose hypotension:** more common in patients taking diuretics

Cautions and contraindications:

- 1) **pregnancy** and **breastfeeding** - **avoid**
- 2) **renovascular disease** - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis
- 3) **aortic stenosis** - may result in hypotension
- 4) patients receiving **high-dose diuretic therapy** (more than 80 mg of furosemide a day) - significantly increases the risk of hypotension
- 5) hereditary of idiopathic **angioedema**

Monitoring:

- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
- A rise in the creatinine and potassium may be expected after starting ACE inhibitors.
- Acceptable changes are an increase in serum creatinine, up to **30%** from baseline and an increase in potassium up to **5.5 mmol/l**

The NICE CKD guidelines suggest that a decrease in eGFR of up to 25% or a rise in creatinine of up to 30% is acceptable

Bendroflumethiazide

- Bendroflumethiazide (bendrofluazide) is a thiazide diuretic which works by inhibiting sodium absorption at the beginning of the **distal convoluted tubule (DCT)**.
- Potassium is lost as a result of more sodium reaching the collecting ducts.
- Bendroflumethiazide has a role in the treatment of mild heart failure although loop diuretics are better for reducing overload.
- The main use of bendroflumethiazide was in the management of hypertension but recent NICE guidelines now recommend other thiazide-like diuretics such as indapamide and chlorthalidone.

Common adverse effects:

- 1) dehydration
- 2) postural hypotension
- 3) hyponatraemia, hypokalaemia, hypercalcaemia
- 4) gout
- 5) impaired glucose tolerance
- 6) impotence

Rare adverse effects:

- 1) thrombocytopaenia
- 2) agranulocytosis
- 3) photosensitivity rash
- 4) pancreatitis

Loop diuretics

- Furosemide and bumetanide are loop diuretics that act by inhibiting the Na-K-2Cl cotransporter (NKCC) in the thick ascending limb of the loop of Henle, reducing the absorption of NaCl.
- There are two variants of NKCC; loop diuretics act on NKCC2, which is more prevalent in the kidneys

Indications:

- 1) heart failure: both acute (usually intravenously) and chronic (usually orally)
- 2) resistant hypertension, particularly in patients with renal impairment

Adverse effects:

- 1) hypotension
- 2) hypocalcaemia
- 3) hyponatraemia
- 4) hypokalaemia
- 5) hypochloraemic alkalosis
- 6) ototoxicity
- 7) renal impairment (from dehydration + direct toxic effect)
- 8) hyperglycaemia (less common than with thiazides)
- 9) gout

Spironolactone

Spironolactone is an aldosterone antagonist which acts in the cortical collecting duct.

Indications:

- 1) ascites:
 - ✓ Patients with cirrhosis develop a secondary hyperaldosteronism.
 - ✓ Relatively large doses such as 100 or 200mg are often used
- 2) hypertension: used in some patients as a NICE 'step 4' treatment
- 3) heart failure (see RALES study below)
- 4) nephrotic syndrome
- 5) Conn's syndrome

Adverse effects:

- hyperkalaemia
- gynaecomastia

RALES

- NYHA III + IV, patients already taking ACE inhibitor
- low dose spironolactone reduces all cause mortality

Adrenaline

Adrenaline is a sympathomimetic amine with both alpha and beta adrenergic stimulating properties

Indications:

- anaphylaxis
- cardiac arrest

Recommend Adult Life Support (ALS) adrenaline doses:

- 1) anaphylaxis: 0.5ml 1:1,000 IM
- 2) cardiac arrest:
 - ⇒ 1ml of 1:1000 IV
 - ⇒ 10ml 1:10,000 IV

Management of accidental injection:

- local infiltration of phentolamine

Adrenoceptor antagonists

Alpha antagonists

- alpha-1: doxazosin
- alpha-1a: tamsulosin - acts mainly on **urogenital tract**
- alpha-2: yohimbine
- non-selective: **phenoxybenzamine** (previously used in peripheral arterial disease)

Beta antagonists

- beta-1: **atenolol**
- non-selective: **propranolol**

Mixed alpha and beta antagonists

- **Carvedilol** and **labetalol**

Beta-blocker overdose

Features:

- bradycardia
- hypotension
- heart failure
- syncope

Management:

- if bradycardic then **atropine**
- in resistant cases **glucagon** may be used
- Haemodialysis is not effective in beta-blocker overdose
- Overdose of beta-blockers or calcium channel blocker can lead to significant bradycardia.
- If taken **within one hour** of presentation, **activated charcoal** should be tried.
- If there is symptomatic bradycardia **atropine** should be used **in the first instance**.
- **Glucagon** can be effective but this should be tried after atropine.
- Pacing may be necessary if these drug treatments fail.

NICE guidelines (CG108 Chronic heart failure) maintain that

- **Cardioselective beta-blockers** should be tried in patients with left ventricular systolic dysfunction even if they have a diagnosis of:
 - Chronic obstructive pulmonary disease (COPD)
 - Peripheral vascular disease
 - Diabetes
 - Erectile dysfunction, or
 - Interstitial pulmonary disease.
- Beta-blockers should not be commenced in the setting of acute exacerbations of COPD or cardiac failure.

Calcium channel blockers

- Calcium channel blockers are primarily used in the management of cardiovascular disease.
- Voltage-gated calcium channels are present in:
 - 1) myocardial cells,
 - 2) cells of the conduction system
 - 3) the vascular smooth muscle cells

The various types of calcium channel blockers have varying effects on these three areas and it is therefore important to differentiate their uses and actions.

Examples	Indications & notes	Side-effects and cautions
Verapamil	☒ Angina, hypertension, arrhythmias • Highly negatively inotropic • Should not be given with beta-blockers as may cause heart block • Should not be given in ventricular tachycardia	1) Heart failure, 2) hypotension, bradycardia, 3) flushing 4) constipation
Diltiazem	☒ Angina, hypertension • Less negatively inotropic than verapamil but caution should still be exercised when patients have heart failure or are taking beta-blockers	1) heart failure 2) Hypotension, bradycardia, 3) ankle swelling
Nifedipine, amlodipine, felodipine (dihydropyridines)	☒ Hypertension, angina, Raynaud's • Affects the peripheral vascular smooth muscle more than the myocardium and therefore do not result in worsening of heart failure	1) Flushing, headache, 2) ankle swelling

Digoxin and digoxin toxicity

- A cardiac glycoside now mainly used for rate control in the management of **AF**.
- As it has positive inotropic properties it is sometimes used for improving symptoms (but not mortality) in patients with **heart failure**.

Mechanism of action:

- 1) **decreases conduction** through AVN which slows the ventricular rate in AF and flutter
- 2) Increase the force of cardiac muscle contraction through **inhibition** of the **Na⁺/K⁺ ATPase pump**
- 3) Also **stimulates vagus nerve**

Digoxin toxicity

- Plasma concentration alone does not determine whether a patient has developed toxicity
- The BNF advises that the likelihood of toxicity increases progressively from **1.5 to 3 mcg/l**

Features:

- 1) generally unwell, lethargy, anorexia, nausea, vomiting, confusion, yellow-green vision
- 2) hyperkalaemia, metabolic acidosis,
- 3) Brady- and tachyarrhythmia.
- 4) AV block
- 5) Hypotension can occur due to the negative chronotropic effects of digoxin.

Precipitating factors:

- 1) classically: hypokalaemia, hypomagnesaemia
- 2) hypoalbuminaemia, hypothermia, hypothyroidism
- 3) hypercalcaemia, hypernatraemia,
- 4) increasing age
- 5) renal failure
- 6) acidosis
- 7) myocardial ischaemia
- 8) Drugs:
 - ✓ **Amiodarone, quinidine,**
 - ✓ **verapamil, diltiazem,**
 - ✓ **spironolactone:** competes for secretion in distal convoluted tubule therefore reduce excretion
 - ✓ **Ciclosporin.**
 - ✓ **Drugs which cause hypokalaemia** e.g. thiazides and loop diuretics

*hyperkalaemia may also worsen digoxin toxicity, although this is very small print

Management:

- 1) Digoxin specific antibodies (**Digibind**)
- 2) correct arrhythmias
- 3) monitor potassium

Indications for digoxin specific antibodies:

- 1) **Severe hyperkalaemia** (> 6 mmol/L) resistant to treatment with insulin and dextrose (not calcium gluconate risk of further ventricular arrhythmias)
 - 2) **Bradycardia** unresponsive to atropine with cardiac compromise
 - 3) **Tachycardia** (especially ventricular) associated with cardiac compromise.
- Digibind should be considered at an earlier stage if the patient has pre-existing cardiac disease.
 - In absence of Digibind, insertion of a temporary pacing wire may improve the heart rate.

ECG Features of digoxin

- down-sloping ST depression ('reverse tick')
- flattened/inverted T waves
- short QT interval
- arrhythmias e.g. AV block, Bradycardia

Aspirin

- Aspirin works by blocking the action of both **cyclooxygenase-1** and 2.
- Cyclooxygenase is responsible for synthesis of:
 - 1) **Prostaglandin**,
 - 2) **prostacyclin** and
 - 3) **thromboxane**.
- The blocking of **thromboxane A2** formation in platelets → **reduces the ability of platelets to aggregate** which has led to the widespread use of low-dose aspirin in cardiovascular disease.
- Until recent guidelines changed all patients with established cardiovascular disease took aspirin if there was no contraindication. Following the 2010 technology appraisal of clopidogrel this is no longer the case*.

What do the current guidelines recommend?

- **first-line** for patients with **ischaemic heart disease**

Potentiates

- oral hypoglycaemics
- warfarin
- steroids

Two recent trials (the Aspirin for Asymptomatic Atherosclerosis and the Antithrombotic Trialists Collaboration) have cast doubt on the use of aspirin in primary prevention of cardiovascular disease. Guidelines have not yet changed to reflect this. However the Medicines and Healthcare products Regulatory Agency (MHRA) issued a drug safety update in January 2010 reminding prescribers that **aspirin is not licensed for primary prevention**.

*NICE now recommend clopidogrel first-line following an ischaemic stroke and for peripheral arterial disease. For TIAs the situation is more complex. Recent Royal College of Physician (RCP) guidelines support the use of clopidogrel in TIAs. However the older NICE guidelines still recommend aspirin + dipyridamole - a position the RCP state is 'illogical'

Dipyridamole

Dipyridamole is an antiplatelet mainly used in combination with aspirin after an ischaemic stroke or transient ischaemic attack.

Mechanism of action

- **inhibits phosphodiesterase** → elevating platelet cAMP levels which in turn **reduce intracellular calcium levels**
- other actions include:
 - ⇒ reducing cellular uptake of adenosine and
 - ⇒ inhibition of thromboxane synthase

Cardiac enzymes and protein markers

Interpretation of the various cardiac enzymes has now largely been superseded by the introduction of troponin T and I. Questions still however commonly appear in exams.

Key points for the exam

- **Myoglobin** is the first to rise
- **CK-MB** is useful to look for reinfarction as it returns to normal after 2-3 days (troponin T remains elevated for up to 10 days)

	Begins to rise	Peak value	Returns to normal
Myoglobin	1-2 hours	6-8 hours	1-2 days
CK-MB	2-6 hours	16-20 hours	2-3 days
CK	4-8 hours	16-24 hours	3-4 days
Trop T	4-6 hours	12-24 hours	7-10 days
AST	12-24 hours	36-48 hours	3-4 days
LDH	24-48 hours	72 hours	8-10 days

Elevated troponin:

- Sepsis
- Burns
- Trauma
- Rhabdomyolysis
- Pulmonary embolism
- Pulmonary hypertension
- Hypertension
- Hypotension, especially with arrhythmias
- Hypertrophic obstructive cardiomyopathy
- Cardioversion
- Myocarditis including Kawasaki's disease
- Subarachnoid haemorrhage and stroke
- **TPA** مهمه جدا وخطيرة لانك ممكن تشخصه غلط وتموته لو اديته
- Infiltrative/autoimmune disorders including sarcoidosis, amyloidosis, haemochromatosis and scleroderma.
- Drugs including Adriamycin, Herceptin and 5-fluorouracil

Coronary circulation

Arterial supply of the heart:

- left aortic sinus → left coronary artery (LCA) → LADA + circumflex
- right aortic sinus → right coronary artery (RCA) → posterior descending
- RCA supplies **SA node** in 60%, **AV node** in 90%

Venous drainage of the heart: → coronary sinus drains into the right atrium

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, Avf	Right coronary
Anterolateral	V4-6, I, aVL	Left anterior descending or left circumflex
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Tall R waves V1-2	Usually left circumflex, also right coronary

The peak incidence of **STEMI** and the peak incidence of death due to ischaemic heart disease both coincide at **around 8-9 am**.

The early morning is associated with several physiological and haematological factors which predispose to vasospasm, infarction and death.

There is

- **Increasing adrenergic activity**
- **Increased plasma fibrinogen levels**
- **Increased inhibition of fibrinolysis and**
- **Increased platelet adhesiveness.**

Thus it is likely that a pro-thrombotic, adrenergic environment in the presence of atherosclerosis may proceed to infarction.

Interestingly, NSTEMIs are not associated with this degree of diurnal rhythm.

Precipitating factors for an infarct include:

- Emotional stressors.
- Surgical procedure
- Heavy and Modest physical exertion
- Rest, Sleep

Chest pain

- NICE issued guidelines in 2010 on the 'Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin.'

Patients presenting with stable chest pain:

- NICE guidelines suggest an approach where the risk of a patient having coronary artery disease (CAD) is calculated based on their **symptoms** (whether they have typical angina, atypical angina or non-anginal chest pain), **age**, **gender** and **risk factors**.
- NICE define anginal pain as the following:
 - 1) **Constricting discomfort** in the front of the chest, neck, shoulders, jaw or arms
 - 2) **Precipitated** by physical exertion
 - 3) **Relieved** by rest or GTN in about **5 minutes**
- Patients with all **3** features have **typical angina**
- Patients with **2** of the above features have **atypical angina**
- Patients with **1** or none of the above features have **non-anginal chest pain**
- If patients have **typical anginal symptoms** and a **risk of CAD** is **greater than 90%** then:
 - ⇒ **No further diagnostic testing** is required.
 - ⇒ It should be noted that all men over the age of 70 years who have typical anginal symptoms fall into this category.
- If patients have **typical anginal symptoms** and **estimated risk** of **10-90%**
 - ⇒ The following investigations are recommended. Note the absence of the exercise tolerance test:

Estimated likelihood of CAD	Diagnostic testing
61-90%	Coronary angiography
30-60%	Functional imaging , for example: • stress echocardiography • myocardial perfusion scan with SPECT • first-pass contrast-enhanced magnetic resonance (MR) perfusion • MR imaging for stress-induced wall motion abnormalities.
10-29%	CT calcium scoring

Angina pectoris management:

- The management of stable angina comprises **lifestyle changes**, **medication**, **percutaneous coronary intervention** and **surgery**.
- NICE produced guidelines in 2011 covering the management of stable angina

Medication:

- **all patients should receive aspirin** and a **statin** in the absence of any contraindication
- sublingual **glyceryl trinitrate** to abort angina attacks
- NICE recommend using either a **beta-blocker** or a **calcium channel blocker** **first-line** based on 'comorbidities, contraindications and the person's preference'
- **If a calcium channel blocker is used** as monotherapy → a rate-limiting one such as **verapamil** or **diltiazem** should be used.
- **If calcium channel blocker used in combination with a beta-blocker** then use a long-acting dihydropyridine calcium-channel blocker (e.g. **modified-release nifedipine**).
 - + Remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- **If there is a poor response** to initial treatment then medication should be increased to **the maximum tolerated dose** (e.g. for atenolol 100mg od)
- **If a patient is still symptomatic** after monotherapy with a beta-blocker **add** a calcium channel blocker and vice versa
- **If a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker** then consider one of the following drugs: **a long-acting nitrate**, **ivabradine**, **nicorandil** or **ranolazine**
- **If a patient is taking both a beta-blocker and a calcium-channel blocker** then only add a third drug whilst a patient is awaiting **assessment for PCI or CABG**

Nitrate tolerance:

- many patients who take nitrates develop tolerance and experience reduced efficacy
- The BNF advises that patients who develop tolerance should take the second dose of isosorbide mononitrate after 8 hours, rather than after 12 hours. This allows blood-nitrate levels to fall for 4 hours and maintains effectiveness
- this effect is not seen in patients who take modified release isosorbide mononitrate

Ivabradine

- a new class of anti-anginal drug which works by **reducing the heart rate**
- acts on the I_f ('funny') ion current which is highly expressed in the sinoatrial node, reducing cardiac pacemaker activity

Adverse effects:

- 1) **Visual effects**, particular luminous phenomena, are common.
 - 2) **Bradycardia**, due to the mechanism of action, may also be seen
- there is no evidence currently of superiority over existing treatments of stable angina

Acute coronary syndrome

Patients presenting with acute chest pain

1) Immediate management of suspected acute coronary syndrome (ACS):

- 1) Glyceryl trinitrate
- 2) Aspirin 300mg. NICE do not recommend giving other antiplatelet agents (i.e. Clopidogrel) outside of hospital
- 3) Do not routinely give oxygen, only give if sats < 94%
- 4) Perform an ECG as soon as possible but do not delay transfer to hospital.

A normal ECG does not exclude ACS

2) Referral:

- 1) current chest pain or chest pain in the last 12 hours with an abnormal ECG:
⇒ emergency admission
- 2) chest pain 12-72 hours ago:
⇒ refer to hospital the same-day for assessment
- 3) chest pain > 72 hours ago:
⇒ perform full assessment with ECG and troponin measurement before deciding upon further action

NICE suggest the following in terms of oxygen therapy:

- Do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission.
- Only offer supplemental oxygen to:
 - 1) people with oxygen saturation (SpO₂) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO₂ of 94-98%
 - 2) People with COPD who are at risk of hypercapnic respiratory failure, to achieve a target SpO₂ of 88-92% until blood gas analysis is available.

Management of NSTEMI

- NICE produced guidelines in **2013** on the Secondary prevention in primary and secondary care for patients following a myocardial infarction management of unstable angina and NSTEMI.

1) All patients should receive:

- aspirin 300mg**
- nitrates** or **morphine** to relieve chest pain if required
 - Whilst it is common that non-hypoxic patients receive oxygen therapy there is little evidence to support this approach.
 - The 2008 British Thoracic Society oxygen therapy guidelines advise not giving oxygen unless the patient is hypoxic.

2) Antithrombin treatment:

- Fondaparinux** should be offered to patients who are not at a high risk of bleeding and who are not having angiography within the next 24 hours.
- Unfractionated heparin** should be given:
 - ⇒ If **angiography is likely** within 24 hours or
 - ⇒ a patient's **creatinine > 265 umol/l**

3) Clopidogrel 300mg should be given to **all patients** and continued for **12 months**

4) Intravenous glycoprotein IIb/IIIa receptor antagonists

- Eptifibatide** or **tirofiban**
- It should be given to:
 - Patients who have an **intermediate or higher risk** of adverse cardiovascular events (**predicted 6-month mortality above 3%**), and
 - Who are scheduled to undergo **angiography within 96 hours** of hospital admission

5) Coronary angiography:

- Should be considered **within 96 hours** of first admission to hospital to patients who have a **predicted 6-month mortality above 3%**
- It should also be performed **as soon as possible** in patients who are **clinically unstable**.

Mechanism of action of drugs commonly used in the management of acute coronary syndrome:

Medication	Mechanism of action
Aspirin	Antiplatelet - inhibits the production of thromboxane A2
Clopidogrel	Antiplatelet - inhibits ADP binding to its platelet receptor
Enoxaparin & Fondaparinux	Activates antithrombin III , which in turn potentiates the inhibition of coagulation factors Xa
Bivalirudin	Reversible direct thrombin inhibitor

Management STEMI

1) **In the absence of contraindications, all patients should be given:**

- ☒ Aspirin
- ☒ clopidogrel: the two major studies (CLARITY and COMMIT) both confirmed benefit but used different loading doses (300mg and 75mg respectively)
- ☒ LMWH

NICE suggest the following in terms of oxygen therapy:

- Do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission.
- Only offer supplemental oxygen to:
 - people with oxygen saturation (SpO₂) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO₂ of **94-98%**
 - People with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO₂ of **88-92%** until blood gas analysis is available.

2) **Primary percutaneous coronary intervention (PCI)**

has emerged as the **gold-standard treatment** for STEMI but is not available in all centres.

3) **Thrombolysis** should be performed in patients without access to primary PCI

- ☒ **tissue plasminogen activator (tPA)** has been shown to offer **clear mortality benefits** over **streptokinase**
 - ⇒ **tenecteplase** is **easier to administer** and has been shown to have non-inferior efficacy to **alteplase** with a similar adverse effect profile
- ☒ An **ECG** should be performed **90 minutes** following thrombolysis to assess whether there has been a greater than **50% resolution** in the **ST elevation**
 - if there has **not been adequate resolution** then **rescue PCI** is superior to repeat thrombolysis
 - For patients **successfully treated with thrombolysis** **PCI has been shown to be beneficial**. The optimal timing of this is still under investigation

Glycaemic control in patients with diabetes mellitus:

- in 2011 NICE issued guidance on the management of hyperglycaemia in acute coronary syndromes
 - ⇒ it recommends using a dose-adjusted insulin infusion with regular monitoring of blood glucose levels to glucose below 11.0 mmol/l
 - ⇒ intensive insulin therapy (an intravenous infusion of insulin and glucose with or without potassium, sometimes referred to as 'DIGAMI') regimes are not recommended routinely

Percutaneous coronary intervention (PCI)

- PCI is a technique used to **restore myocardial perfusion** in patients with ischaemic heart disease, both in patients with stable angina and acute coronary syndromes.
- **Stents are implanted** in around 95% of patients - it is now rare for just balloon angioplasty to be performed
- **Following stent insertion;**
 - ⇒ **Migration and proliferation of smooth muscle cells and fibroblasts** occur to the treated segment.
 - ⇒ The stent struts eventually become covered by endothelium.
 - ⇒ Until this happens there is an increased risk of platelet aggregation leading to thrombosis.

2 main complications may occur:

1) Stent thrombosis:

- Due to **platelet aggregation** as above.
- Occurs in 1-2% of patients,
- Most commonly in the **first month**.
- Usually presents with **acute myocardial infarction**

2) Restenosis:

- Due to **excessive tissue proliferation around stent**.
- Occurs in around 5-20% of patients,
- Most commonly in the first **3-6 months**.
- Usually presents with the **recurrence of angina symptoms**.
- Risk factors include **diabetes**, **renal impairment** and **stents in venous** bypass grafts

Types of stent:

- **bare-metal stent (BMS)**
- **Drug-eluting stents (DES):**
 - ✓ Stent coated with **paclitaxel** or **rapamycin** which inhibit local tissue growth.
 - ✓ This **reduces restenosis** rates
 - ✓ The stent **thrombosis rates are increased** as the process of stent endothelialisation is slowed
- Following insertion the most important factor in **preventing stent thrombosis** is **antiplatelet therapy**.
- Aspirin should be **continued indefinitely**.
- The length of **clopidogrel** treatment depends on the type of stent, reason for insertion and consultant preference

Thrombolysis

- Thrombolytic drugs activate plasminogen to form plasmin.
- This in turn degrades fibrin and help breaks up thrombi.
- They in primarily used in patients who present with a ST elevation myocardial infarction.
- Other indications include acute ischaemic stroke and pulmonary embolism, although strict inclusion criteria apply.

Examples:

- alteplase
- tenecteplase
- streptokinase

Contraindications to thrombolysis:

- 1) active internal bleeding
- 2) recent haemorrhage, trauma or surgery (including dental extraction)
- 3) coagulation and bleeding disorders
- 4) intracranial neoplasm
- 5) stroke < 3 months
- 6) recent head injury
- 7) aortic dissection
- 8) pregnancy
- 9) severe hypertension

Side-effects:

- 1) haemorrhage
- 2) hypotension - more common with streptokinase
- 3) allergic reactions may occur with streptokinase

Secondary prevention of Myocardial infarction:

- NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2013.

1) All patients should be offered the following drugs:

1. **Dual antiplatelet therapy** (aspirin plus a second antiplatelet agent)
2. **ACE inhibitor**
3. **Beta-blocker**
4. **Statin**

2) Some selected lifestyle points:

Diet: advise a Mediterranean style diet, switch butter and cheese for plant oil based products. Do not recommend omega-3 supplements or eating oily fish

Exercise: advise 20-30 mins a day until patients are slightly breathless

Sexual activity

- ✓ May resume **4 weeks** after an uncomplicated MI.
- ✓ Reassure patients that sex does not increase their likelihood of a further MI.
- ✓ PDE5 inhibitors (e.g, sildenafil) may be used **6 months** after a MI.
- ✓ They should however be avoided in patient prescribed either nitrates or nicorandil

Aldosterone antagonists:

- Patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction;
⇒ treatment with an aldosterone antagonist licensed for post-MI treatment (e.g. **eplerenone**) should be initiated within 3-14 days of the MI, preferably after ACE inhibitor therapy

Prognostic factors:

The 2006 Global Registry of Acute Coronary Events (GRACE) study has been used to derive regression models to predict death in hospital and death after discharge in patients with acute coronary syndrome

Poor prognostic factors:

- 1) age
- 2) development (or history) of heart failure
- 3) peripheral vascular disease
- 4) reduced systolic blood pressure
- 5) Killip class*
- 6) initial serum creatinine concentration
- 7) elevated initial cardiac markers
- 8) cardiac arrest on admission
- 9) ST segment deviation

Killip class - system used to stratify risk **post myocardial infarction**

Killip class	Features	30 day mortality
I	No clinical signs heart failure	6%
II	Lung crackles, S3	17%
III	Frank pulmonary oedema	38%
IV	Cardiogenic shock	81%

Clopidogrel

- An antiplatelet agent used in the management of cardiovascular disease.
- It was previously used when aspirin was not tolerated or contraindicated but there are now a number of conditions for which clopidogrel is used in addition to aspirin, for example in patients with an acute coronary syndrome.
- Following the 2010 NICE technology appraisal clopidogrel is also now first-line in patients following an ischaemic stroke and in patients with peripheral arterial disease.
- Clopidogrel belongs to a class of drugs known as thienopyridines which have a similar mechanism of action.

Other examples include:

- Prasugrel
- Ticagrelor
- Ticlopidine

Mechanism:

- antagonist of the P2Y₁₂ adenosine diphosphate (ADP) receptor, inhibiting the activation of platelets

Interactions:

- Concurrent use of proton pump inhibitors (PPIs) may make clopidogrel less effective (MHRA July 2009)
- This advice was updated by the MHRA in April 2010, evidence seems inconsistent but omeprazole and esomeprazole still cause for concern.
- Other PPIs such as lansoprazole should be OK.

Clopidogrel:

Since clopidogrel came off patent it is now much more widely used post-MI

STEMI:

- ⇒ The European Society of Cardiology recommend dual antiplatelets for 12 months.
- ⇒ In the UK this means aspirin + clopidogrel

NSTEMI

Following the NICE2013 Secondary prevention in primary and secondary care for patients following a myocardial infarction guidelines clopidogrel should be given for the first 12 months

Myocardial infarction complications:

Patients are at risk of a number of immediate, early and late complications following a MI.

Cardiac arrest:

- This most commonly occurs due to patients developing **ventricular fibrillation** and is **the most common cause of death** following a MI.
- Patients are managed as per the ALS protocol with **defibrillation**.

Cardiogenic shock:

- If a **large part of the ventricular myocardium is damaged** in the infarction the ejection fraction of the heart may decrease to the point that the patient develops cardiogenic shock.
- This is difficult to treat.
- Other causes of cardiogenic shock include the 'mechanical' complications such as **left ventricular free wall rupture**.
- Patients may require **inotropic** support and/or an **intra-aortic balloon pump**.

Chronic heart failure:

- As described above, if the patient survives the acute phase their **ventricular myocardium may be dysfunctional** resulting in chronic heart failure.
- **Loop diuretics** such as furosemide will decrease fluid overload.
- Both **ACE-inhibitors** and **beta-blockers** have been shown to improve the long-term prognosis of patients with chronic heart failure.

Tachyarrhythmias:

- **Ventricular fibrillation**, as mentioned above, is the most common cause of death following a MI.
- Other common arrhythmias including **ventricular tachycardia**.

Bradyarrhythmias:

- **Atrioventricular block** is more common following **inferior myocardial infarctions**.

Pericarditis:

- Pericarditis in the **first 48 hours** following a transmural MI is common (10% of patients).
- **The pain** is typical for pericarditis (worse on lying flat etc),
- **a pericardial rub** may be heard and
- **a pericardial effusion** may be demonstrated with an echocardiogram

Dressler's syndrome:

- Tends to occur around **2-6 weeks** following a MI.
- The underlying pathophysiology is thought to be **an autoimmune reaction** against antigenic proteins formed as the myocardium recovers.
- It is characterised by a combination of **fever, pleuritic pain, pericardial effusion and a raised ESR.**
- It is treated with **NSAIDs.**

Left ventricular aneurysm:

- **The ischaemic damage** sustained may **weaken the myocardium** resulting in **aneurysm formation.**
- This is typically associated with **persistent ST elevation** and **left ventricular failure.**
- **Thrombus** may form within the aneurysm increasing the risk of stroke.
- Patients are therefore **anticoagulated.**

Left ventricular free wall rupture:

- This is seen in around **3%** of MIs and occurs around **1-2 weeks** afterwards.
- Patients present with **acute heart failure** secondary to **cardiac tamponade** (**raised JVP, pulsus paradoxus, diminished heart sounds**).
- **Urgent pericardiocentesis** and **thoracotomy** are required.

Ventricular septal defect:

- **Rupture of the interventricular septum** usually occurs in the **first week** and is seen in around **1-2%** of patients.
- Features: **acute heart failure** associated with a **pan-systolic murmur.**
- An **echocardiogram** is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion.
- **Urgent surgical correction** is needed.

Acute mitral regurgitation:

- More common with **infero-posterior infarction** and may be due to **ischaemia** or **rupture of the papillary muscle.**
- An **early-to-mid systolic murmur** is typically heard.
- Patients are treated with **vasodilator therapy** but often require **emergency surgical repair.**

Exercise tolerance tests (ETT, exercise ECG)

Indications:

(ETT has a sensitivity of around 80% and a specificity of 70% for ischaemic heart disease)

- 1) **assessing patients with suspected angina** - however the 2010 NICE Chest pain of recent onset guidelines do not support the use of ETTs for all patients
- 2) **risk stratifying patients** following a myocardial infarction
- 3) **assessing** exercise tolerance
- 4) risk stratifying patients with **hypertrophic cardiomyopathy** (HOCM)

Heart rate:

- **maximum predicted heart rate** = **220 - patient's age**
- **the target heart rate** is **at least 85%** of maximum predicted to allow reasonable interpretation of a test as low-risk or negative

Contraindications:

- 1) **myocardial infarction** less than **7 days** ago
- 2) **unstable angina**
- 3) **uncontrolled HTN** (systolic BP > 180 mmHg) or hypotension (systolic BP < 90 mmHg)
- 4) **aortic stenosis**
- 5) **LBBB**: this would make the ECG very difficult to interpret

Stop if:

- 1) exhaustion / patient request
- 2) 'severe', 'limiting' chest pain
- 3) Stop if rapid ST elevation and pain
- 4) > 2mm ST elevation.
- 5) > 3mm ST depression
- 6) systolic blood pressure > 230 mmHg
- 7) systolic blood pressure falling > 20 mmHg
- 8) heart rate falling > 20% of starting rate
- 9) attainment of maximum predicted heart rate
- 10) arrhythmia develops

Exercise: physiological changes

Blood pressure:

- systolic increases, diastolic decreases
- leads to increased pulse pressure
- in healthy young people the increase in MABP is only slight

Cardiac output:

- increase in cardiac output may be 3-5 fold
- results from venous constriction, vasodilation and increased myocardial contractibility, as well as from the maintenance of right atrial pressure by an increase in venous return
- HR up to 3-fold increase
- SV up to 1.5-fold increase

Statins

Statins **inhibit HMG-CoA reductase**, the rate-limiting enzyme in hepatic cholesterol synthesis

Adverse effects:

1) Myopathy:

- Includes myalgia, myositis, rhabdomyolysis and asymptomatic raised creatine kinase.
- Risks factors for myopathy include advanced age, female sex, low body mass index and presence of multisystem disease such as diabetes mellitus.
- Myopathy is more common in lipophilic statins (**simvastatin**, **atorvastatin**) than relatively hydrophilic statins (rosuvastatin, pravastatin, fluvastatin)

2) Liver impairment:

- The 2014 NICE guidelines recommend checking LFTs at **baseline**, **3 months** and **12 months**.
- Treatment should be discontinued if serum transaminase concentrations rise to and persist at **3 times** the upper limit of the reference range

3) Intracerebral haemorrhage:

- There is some evidence that statins may increase the risk of **intracerebral haemorrhage** in patients who've previously had a stroke.
- This effect is not seen in primary prevention.
- For this reason the Royal College of Physicians recommend **avoiding statins in patients with a history of intracerebral haemorrhage**

Who should receive a statin?

- 1) **All people with established CVD** (stroke, TIA, IHD, peripheral arterial disease)
- 2) NICE 2014 update recommend **anyone with a 10-year cardiovascular risk $\geq 10\%$**
- 3) Patients with type 2 DM should now be **assessed using QRISK2** like other patients are, to determine whether they should be started on statins

Statins should be taken **at night** as this when the majority of cholesterol synthesis occur
This is especially true for **simvastatin** which has a shorter half-life than other statins

☒ NICE guidelines on Cardiovascular disease (CG181) recommend aiming for;

- ⇒ Cholesterol <4 mmol/L and
- ⇒ LDL-C <2 mmol/L in diabetic patients.

Nicotinic acid

- Used in the treatment of patients with hyperlipidaemia, although its use is limited by side-effects.
- As well as lowering cholesterol and triglyceride concentrations it also raises HDL levels

Adverse effects:

- 1) flushing
- 2) impaired glucose tolerance
- 3) myositis

Myocarditis

Causes:

- viral: coxsackie, HIV
- bacteria: diphtheria, clostridia
- spirochaetes: Lyme disease
- protozoa: Chagas' disease, toxoplasmosis
- autoimmune
- drugs: doxorubicin, trastuzumab

Presentation:

- usually young patient with acute history
- chest pain, SOB,

Cardiac tamponade

Features:

- dyspnoea
- **raised JVP, with an absent Y descent** - this is due to the limited right ventricular filling
- tachycardia
- hypotension
- muffled heart sounds
- **pulsus paradoxus**
- **Kussmaul's sign** (much debate about this)
- ECG: electrical alternans

Pulsus paradoxus:

- greater than the normal (10 mmHg) **fall in systolic blood pressure** during **inspiration** → **faint** or **absent pulse** in inspiration
- **severe asthma, cardiac tamponade**

Kussmaul's sign: Paradoxical rise in JVP during inspiration

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

The key differences between constrictive pericarditis and cardiac tamponade are summarised in the table below:

	Cardiac tamponade	Constrictive pericarditis
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present
Characteristic features		Pericardial calcification on CXR



The right sided failure, ascites and pericardial calcification on x ray suggest a diagnosis of constrictive pericarditis.

Cardiomyopathy

Restrictive cardiomyopathy:

Features:

- similar to **constrictive Pericarditis**
- Features suggesting **restrictive cardiomyopathy** rather than constrictive pericarditis
 - 1) prominent apical pulse
 - 2) absence of pericardial calcification on CXR
 - 3) heart may be enlarged
 - 4) ECG abnormalities e.g. bundle branch block, Q waves

Causes:

- 1) amyloidosis (e.g. secondary to myeloma) - **most common cause in UK**
- 2) haemochromatosis
- 3) sarcoidosis
- 4) Loffler's syndrome
- 5) scleroderma

Dilated cardiomyopathy

- dilated heart leading to **systolic dysfunction** (+/- diastolic)
- all 4 chambers affected but LV more so than RV
- absence of congenital, valvular or ischaemic heart disease

Features include:

- 1) **arrhythmias, emboli,**
- 2) **mitral regurgitation**

Causes often considered separate entities:

- 1) alcohol: may improve with **thiamine**
- 2) postpartum
- 3) hypertension

Other causes:

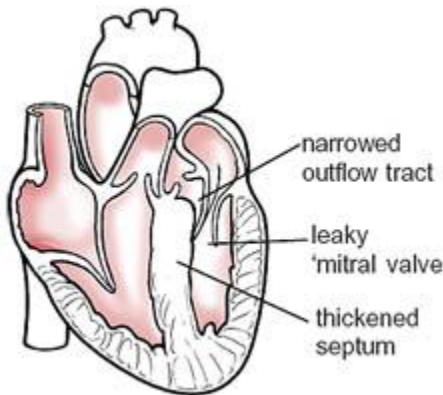
- 1) inherited (see below)
- 2) Coxsackie B, HIV, diphtheria, parasitic
- 3) Hyperthyroidism
- 4) Infiltrative: Haemochromatosis, sarcoidosis (may also lead to restrictive cardiomyopathy)
- 5) neuromuscular e.g. Duchenne muscular dystrophy
- 6) nutritional e.g. Kwashiorkor, pellagra, thiamine/selenium deficiency
- 7) drugs e.g. Doxorubicin

Inherited dilated cardiomyopathy:

- around **1/3 of patients** with DCM are thought to have a genetic predisposition
- a large number of heterogeneous defects have been identified
- the majority of defects are inherited in an **autosomal dominant** fashion although other patterns of inheritance are seen

Hypertrophic obstructive cardiomyopathy (HOCM)

- an **autosomal dominant** disorder of muscle tissue
- Caused by **defects in the genes encoding contractile proteins**.
- The most common defects involve a mutation in the gene encoding:
 - 1) **β -myosin heavy chain protein** or
 - 2) **Myosin binding protein C**.
 - 3) **alpha-tropomyosin** and **troponin T** have been identified.
- The estimated prevalence is **1 in 500**.
- It is an important cause of **sudden death** in apparently healthy individuals.



Features: **often asymptomatic**

- 1) **Septal hypertrophy** causes left ventricular outflow obstruction
- 2) dyspnoea, angina, **syncope**
- 3) **sudden death** (most commonly due to ventricular arrhythmias),
- 4) arrhythmias, heart failure
- 5) jerky pulse, **large 'a' waves**, **double apex beat**
- 6) **ejection systolic murmur**: increases with Valsalva manoeuvre and
Decreases on squatting

Valsalva → ↑ intrathoracic pressure → vein compression → ↓ venous return
Squatting → blood in the leg forced back toward the heart → ↑ venous return
Most murmurs increased with increase venous return except MV prolapse and HOCM

Associations:

- **Friedreich's ataxia**
- **Wolff-Parkinson White**

Echo - mnemonic - **MR SAM ASH**

- mitral regurgitation (**MR**)
- systolic anterior motion (**SAM**) of the anterior mitral valve leaflet
- asymmetric hypertrophy (**ASH**)

ECG:

- LVH
- progressive T wave inversion
- deep Q waves
- atrial fibrillation may occasionally be seen



ECG showing typical changes of HOCM including LVH and T wave inversion

Management:

- Amiodarone
- Beta-blockers or verapamil for symptoms
- Cardioverter defibrillator
- Dual chamber pacemaker
- Endocarditis prophylaxis*

*although see the 2008 NICE guidelines on infective endocarditis prophylaxis

Drugs to avoid:

- nitrates
- ACE inhibitors
- inotropes

Poor prognostic factors

- syncope
- family history of sudden death
- young age at presentation
- non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring
- abnormal blood pressure changes on exercise
- An increased septal wall thickness is also associated with a poor prognosis.

Brugada syndrome

- Inherited cardiovascular disease with may present with **sudden cardiac death**.
- **autosomal dominant**
- Has an estimated prevalence of **1:5,000-10,000**.
- Brugada syndrome is more common in **Asians**.

Pathophysiology:

- a large number of variants exist
- around 20-40% of cases are caused by a mutation in the SCN5A gene which encodes the **myocardial sodium ion channel protein**

ECG changes:

- **convex ST segment elevation** > 2mm in > 1 of V1-V3 followed by a **negative T wave**
- partial right bundle branch block
- changes may be more apparent following **flecainide**



ECG showing Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave

Management:

- **ICD**



Arrhythmogenic right ventricular cardiomyopathy (ARVC)

- also known as arrhythmogenic right ventricular **dysplasia** (ARVD)
- A form of inherited cardiovascular disease which may present with **syncope** or **sudden cardiac death**.
- inherited in an **autosomal dominant** pattern with variable expression
- It is generally regarded as the **second** most common cause of sudden cardiac death in the young after **hypertrophic cardiomyopathy**.

Pathophysiology:

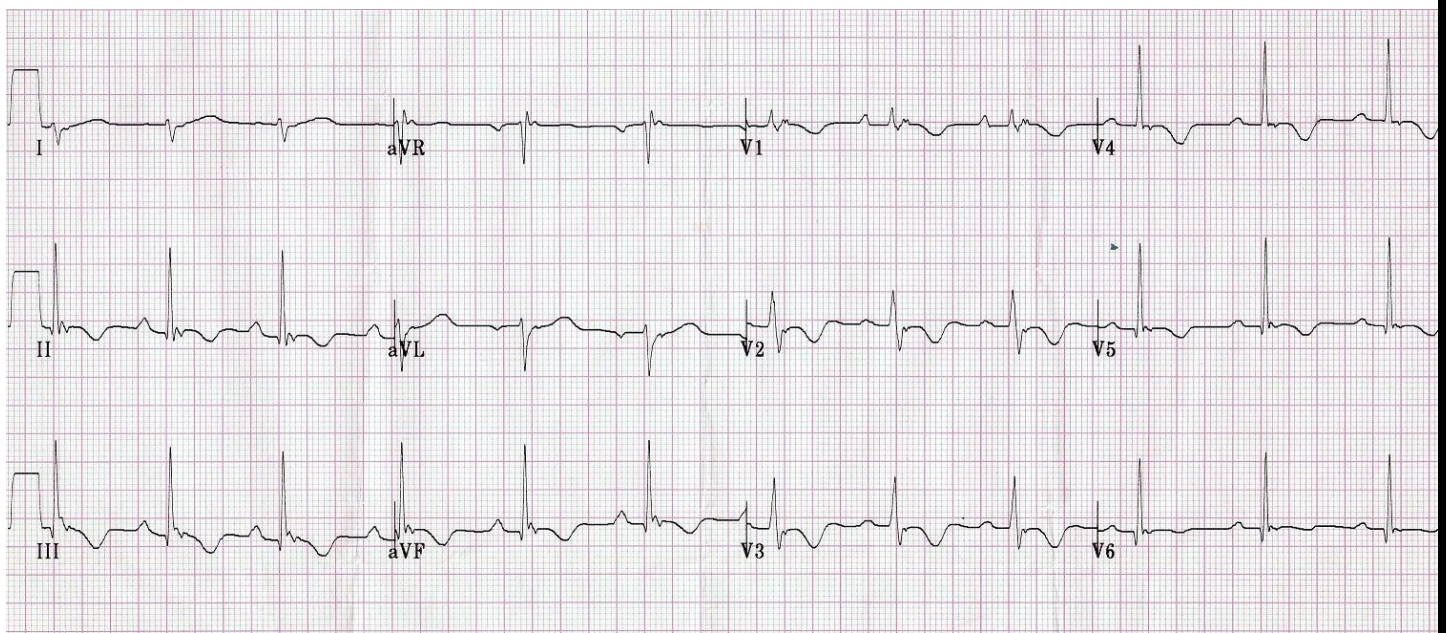
- the right ventricular myocardium is replaced by **fibrofatty tissue**

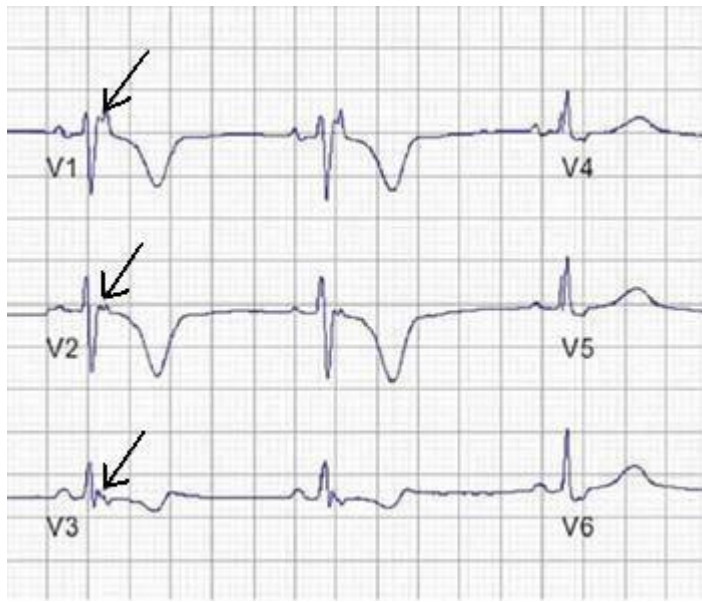
Presentation:

- palpitations
- syncope
- sudden cardiac death

Investigation:

- **ECG abnormalities:**
 - ✓ In V1-3, typically **T wave inversion**.
 - ✓ **An epsilon wave** is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- **Echo changes** are often **subtle** in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- **Magnetic resonance imaging** is useful to show **fibrofatty tissue**





A post-excitation epsilon wave (arrows) in right precordial leads

Management:

- 1) **Sotalol** is the most widely used antiarrhythmic
- 2) **catheter ablation** to prevent ventricular tachycardia
- 3) **ICD**

Naxos disease:

- an **autosomal recessive** variant of ARVC
- a triad of ARVC, **palmoplantar keratosis**, and **woolly hair**



Catecholaminergic polymorphic ventricular tachycardia (CPVT)

- A form of inherited cardiac disease associated with sudden cardiac death.
- It is inherited in an **autosomal dominant** fashion and
- Has a prevalence of around 1:10,000.

Pathophysiology:

- the most common cause is a defect in the **ryanodine receptor** (RYR2) which is found in the myocardial sarcoplasmic reticulum

Features:

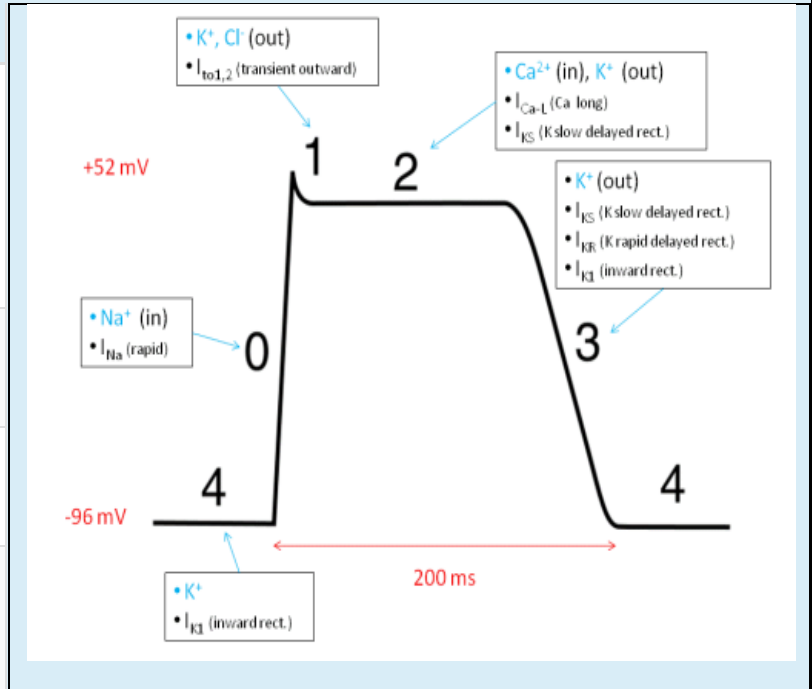
- 1) **exercise or emotion** induced polymorphic ventricular tachycardia resulting in syncope
- 2) sudden cardiac death
- 3) symptoms generally develop before the age of **20 years**

Management:

- 1) **beta-blockers**
- 2) **ICD**

Cardiac action potential

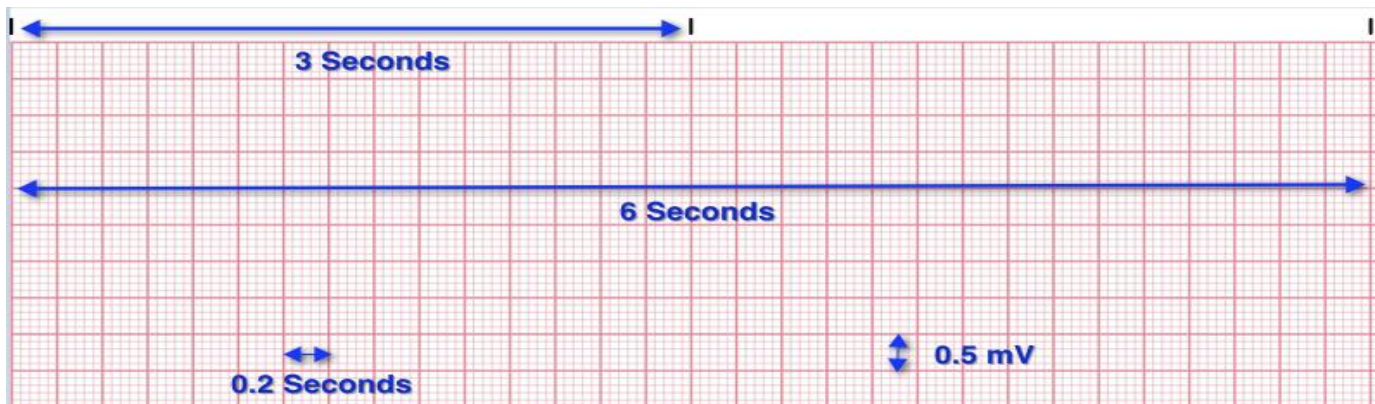
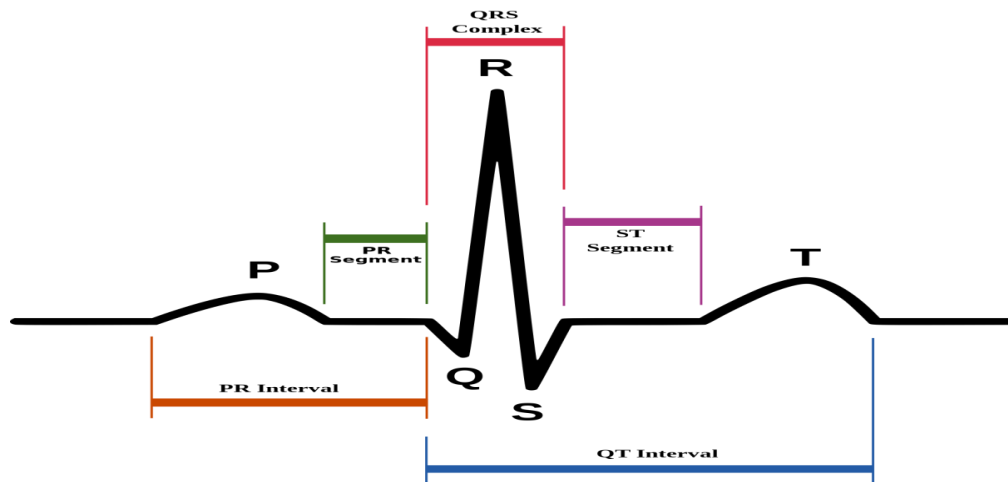
Phase	Description	Mechanism
0	Rapid depolarization	Rapid sodium influx These channels automatically deactivate after a few ms
1	Early repolarisation	Efflux of potassium
2	Plateau	Slow influx of calcium
3	Final repolarisation	Efflux of potassium
4	Restoration of ionic concentrations	- Resting potential is restored by Na^+/K^+ ATPase - There is slow entry of Na^+ into the cell decreasing the potential difference until the threshold potential is reached, triggering a new action potential



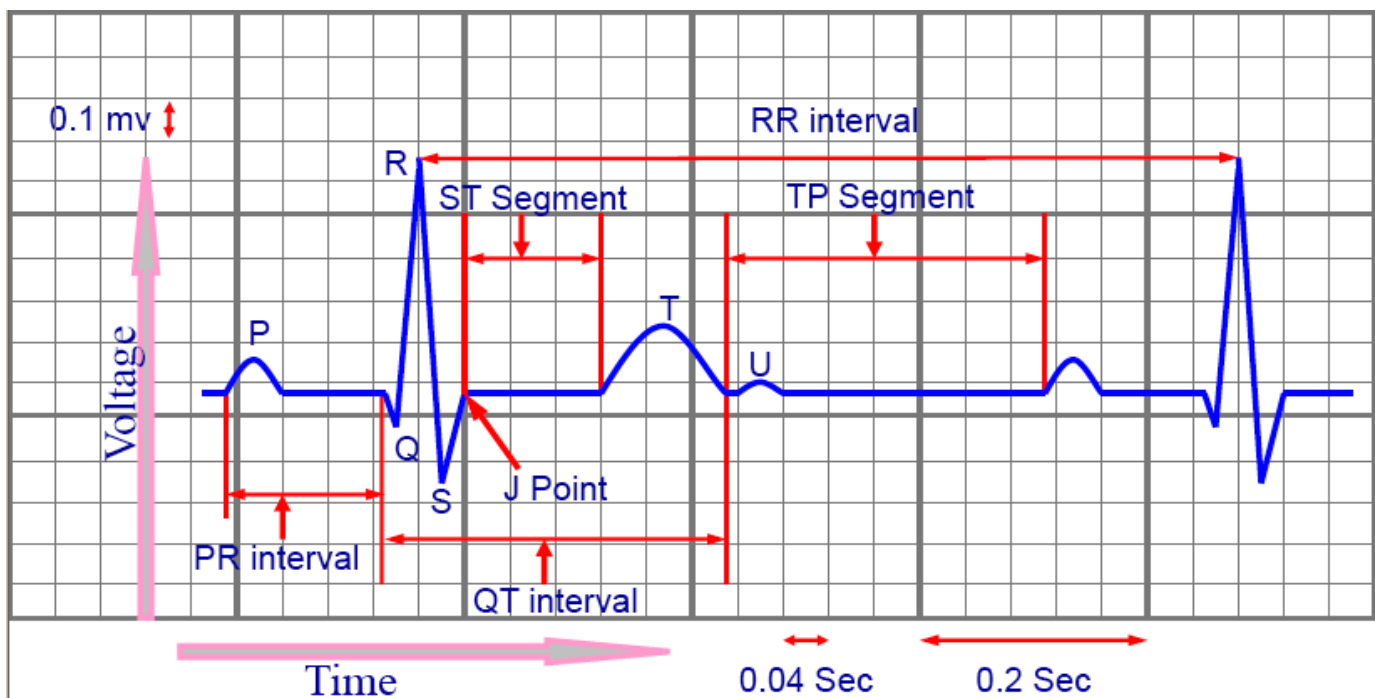
NB cardiac muscle remains contracted 10-15 times longer than skeletal muscle

Conduction velocity

Site	Speed
Atrial conduction	Spreads along ordinary atrial myocardial fibres at 1 m/sec
AV node conduction	0.05 m/sec
Ventricular conduction	Purkinje fibres are of large diameter and achieve velocities of 2-4 m/sec (this allows a rapid and coordinated contraction of the ventricles)



Sec = 1000 msec



- PR interval 0.12 – 0.20 sec
- QRS duration 0.08 – 0.10 sec

- QT interval 0.4 – 0.43 sec
- RR interval 0.6 – 1.0 sec

Arrhythmia

ECG: normal variants in an athlete:

- 1) Sinus bradycardia
- 2) Junctional rhythm
- 3) First degree heart block
- 4) Wenckebach phenomenon

PR interval

1) Causes of a **prolonged** PR interval:

- 1) idiopathic
- 2) ischaemic heart disease
- 3) digoxin toxicity
- 4) hypokalaemia*
- 5) rheumatic fever
- 6) aortic root pathology e.g. abscess secondary to endocarditis
- 7) **Lyme disease**
- 8) sarcoidosis
- 9) myotonic dystrophy
- 10) A prolonged PR interval may also be seen in athletes

*hyperkalaemia can rarely cause a prolonged PR interval, but this is a much less common association than hypokalaemia

2) A **short** PR interval is seen in **Wolff-Parkinson-White syndrome**

ST depression

- 1) secondary to abnormal QRS (LVH, LBBB, RBBB)
- 2) ischaemia
- 3) digoxin
- 4) hypokalaemia
- 5) syndrome X

ST elevation

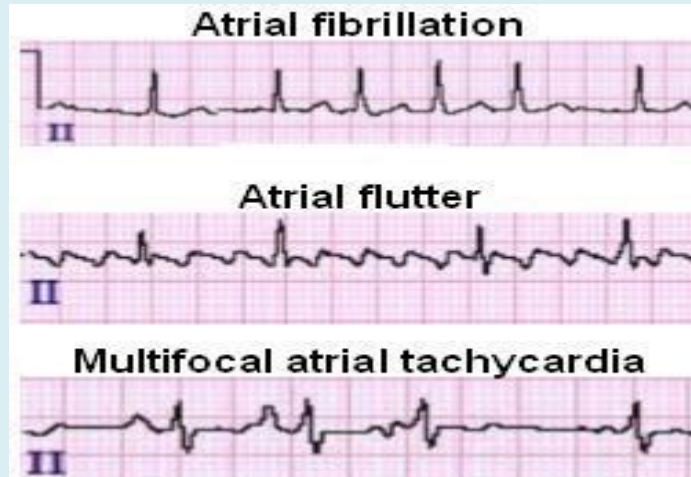
- 1) myocardial infarction
- 2) pericarditis
- 3) left ventricular aneurysm
- 4) normal variant - 'high take-off'
- 5) Prinzmetal's angina (coronary artery spasm)
- 6) rare: **subarachnoid haemorrhage** **tPA** ممكن تموت العيان لو اديته
- 7) Part of spectrum of changes in hyperkalaemia

Multifocal atrial tachycardia (MAT)

- May be defined as an irregular cardiac rhythm caused by at least **three different sites** in the **atria**, which may be demonstrated by **morphologically distinctive P waves**.
- It is more common in **elderly** patients with chronic lung disease, for example **COPD**

Management:

- 1) correction of hypoxia and electrolyte disturbances
- 2) rate-limiting **calcium channel blockers** **Verapamil** are often used **first-line**
- 3) **cardioversion** and **digoxin** are not useful in the management of MAT



Supraventricular tachycardia

- Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of **paroxysmal SVT**.
- Episodes are characterised by the **sudden onset** of a **narrow complex** tachycardia, typically an **atrioventricular nodal re-entry tachycardia (AVNRT)**.
- Other causes include:
 - ✓ **atrioventricular re-entry tachycardias (AVRT)** and
 - ✓ **junctional tachycardias**.

Acute management:

- 1) **Vagal manoeuvres**: e.g. Valsalva manoeuvre
- 2) intravenous **adenosine**
 - ⇒ 6mg → 12mg → 12mg
 - ⇒ contraindicated in asthmatics - **verapamil** is a preferable option
- 3) **Electrical cardioversion**

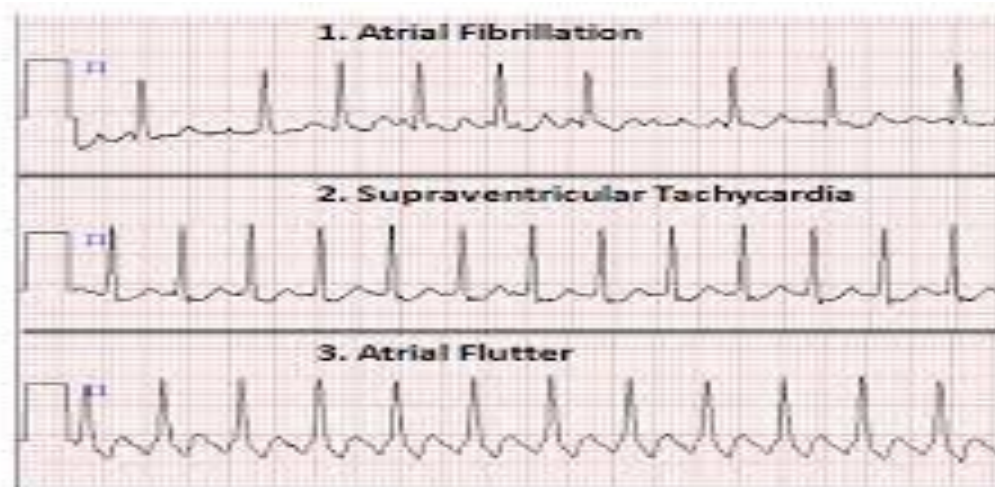
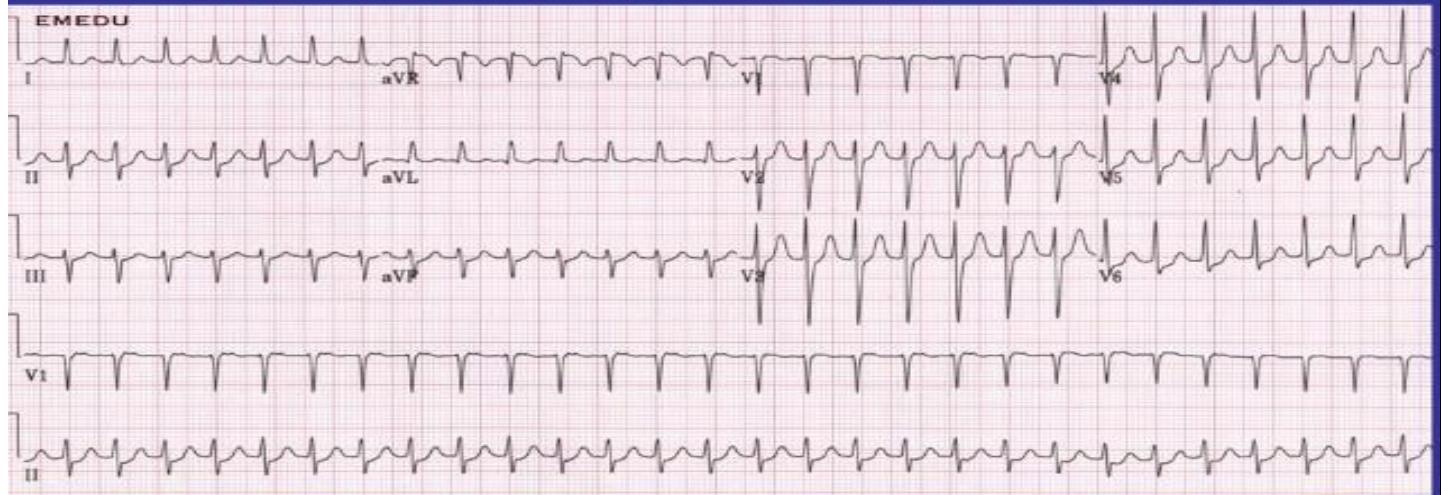
Prevention of episodes:

- 1) **Beta-blockers**
- 2) **Radio-frequency ablation**

SVT + WPW syndrome + asthma

- This patient's SVT should not be terminated with verapamil or digoxin, as these drugs may accelerate anterograde conduction down the accessory pathway (Bundle of Kent).
- In this patient, adenosine is relatively contraindicated with the history of asthma, as it can precipitate an attack.
- Beta-blocker therapy is contraindicated due to the asthma also.
- Amiodarone does not have a place for routinely terminating SVTs, however it can be effective in this situation as long as it is given slowly.
- "Adenosine should be avoided in patients with AV block and asthma, and care should be taken in patients with COPD because it may provoke bronchospasm.
- Verapamil should not be used in the presence of WPW syndrome, previous β -blockade or if signs of impaired left ventricular function exist

Supraventricular Tachycardia



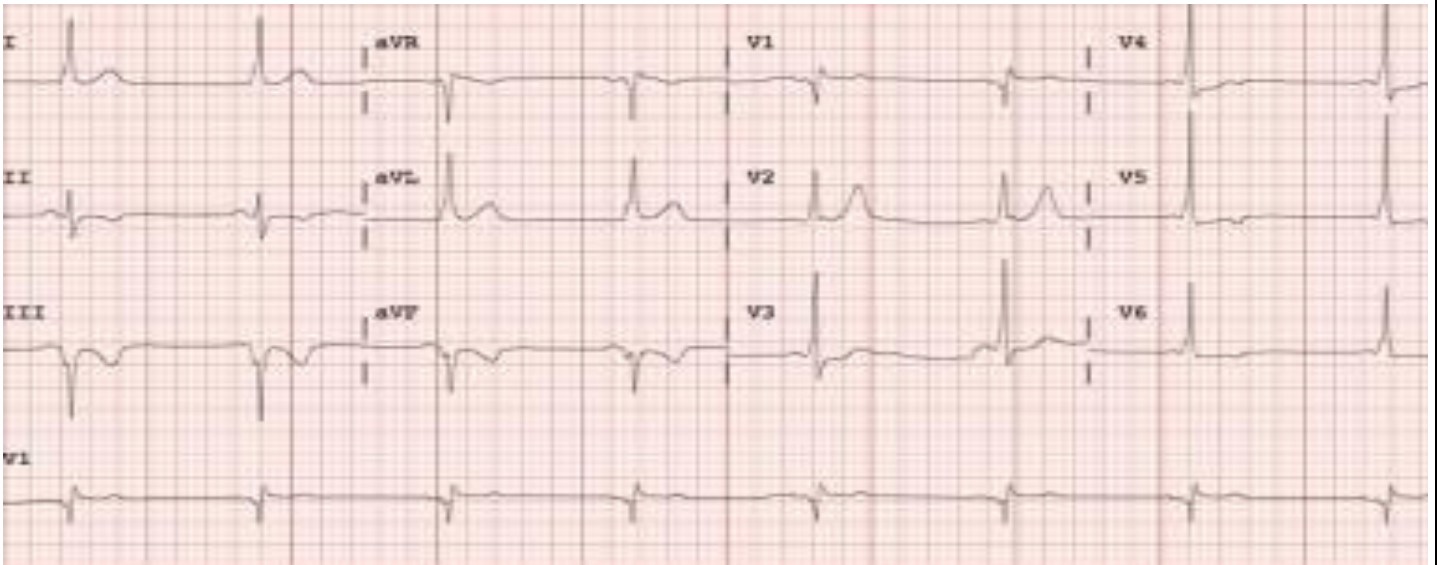
Wolff-Parkinson White (WPW)

- WPW is caused by a **congenital accessory conducting pathway** between the atria and ventricles leading to atrioventricular re-entry tachycardia (**AVRT**).
- As the accessory pathway does not slow conduction AF can degenerate rapidly to VF

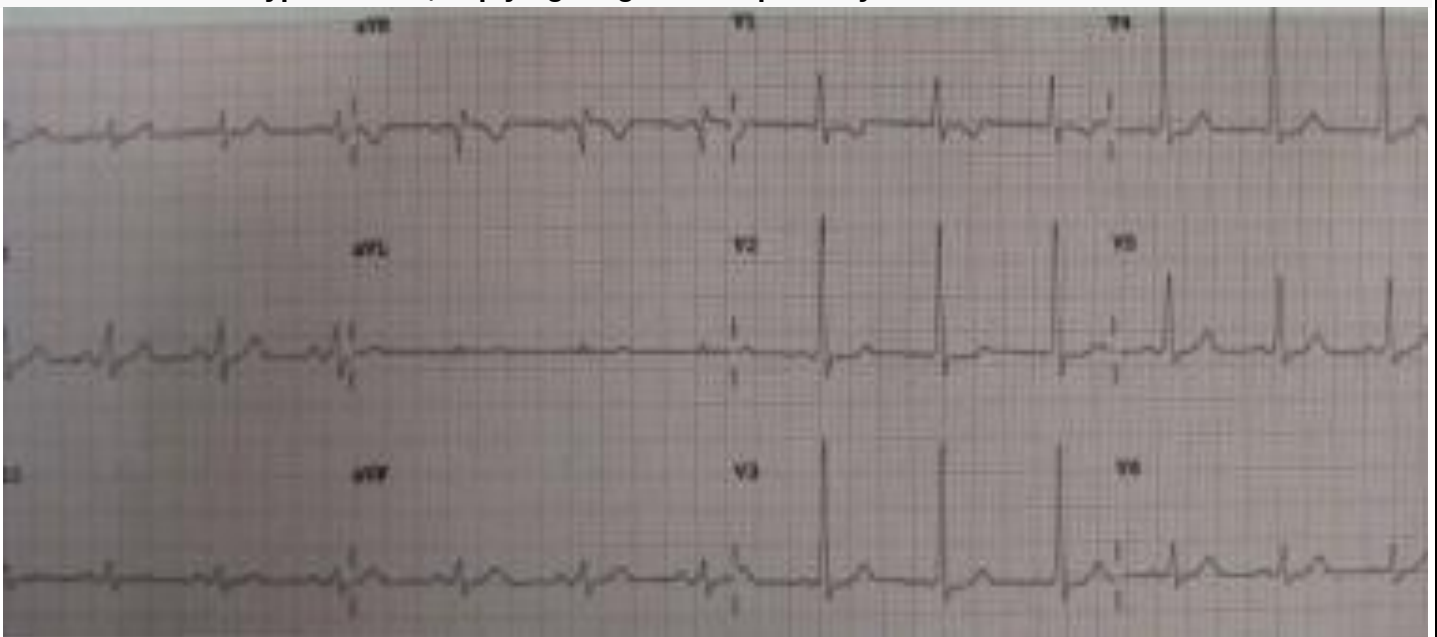
Possible ECG features include:

- 1) **short PR interval**
- 2) **wide QRS** complexes with a **slurred upstroke** - 'delta wave'
- 3) **left axis deviation** if right-sided accessory pathway*
- 4) right axis deviation if left-sided accessory pathway*

***in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation**



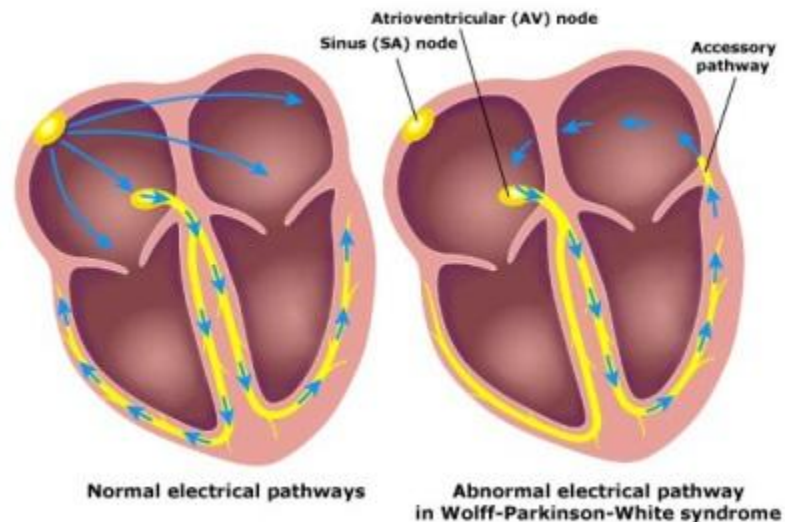
ECG showing short PR interval associated with a slurred upstroke (delta wave). Note the non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia. The left axis deviation means that this is type B WPW, implying a right-sided pathway



Further example showing a characteristic delta wave

Differentiating between type A and type B

- **Type A** (left-sided pathway): **dominant R** wave in **V1**
- **Type B** (right-sided pathway): no dominant R wave in **V1**



Associations of WPW:

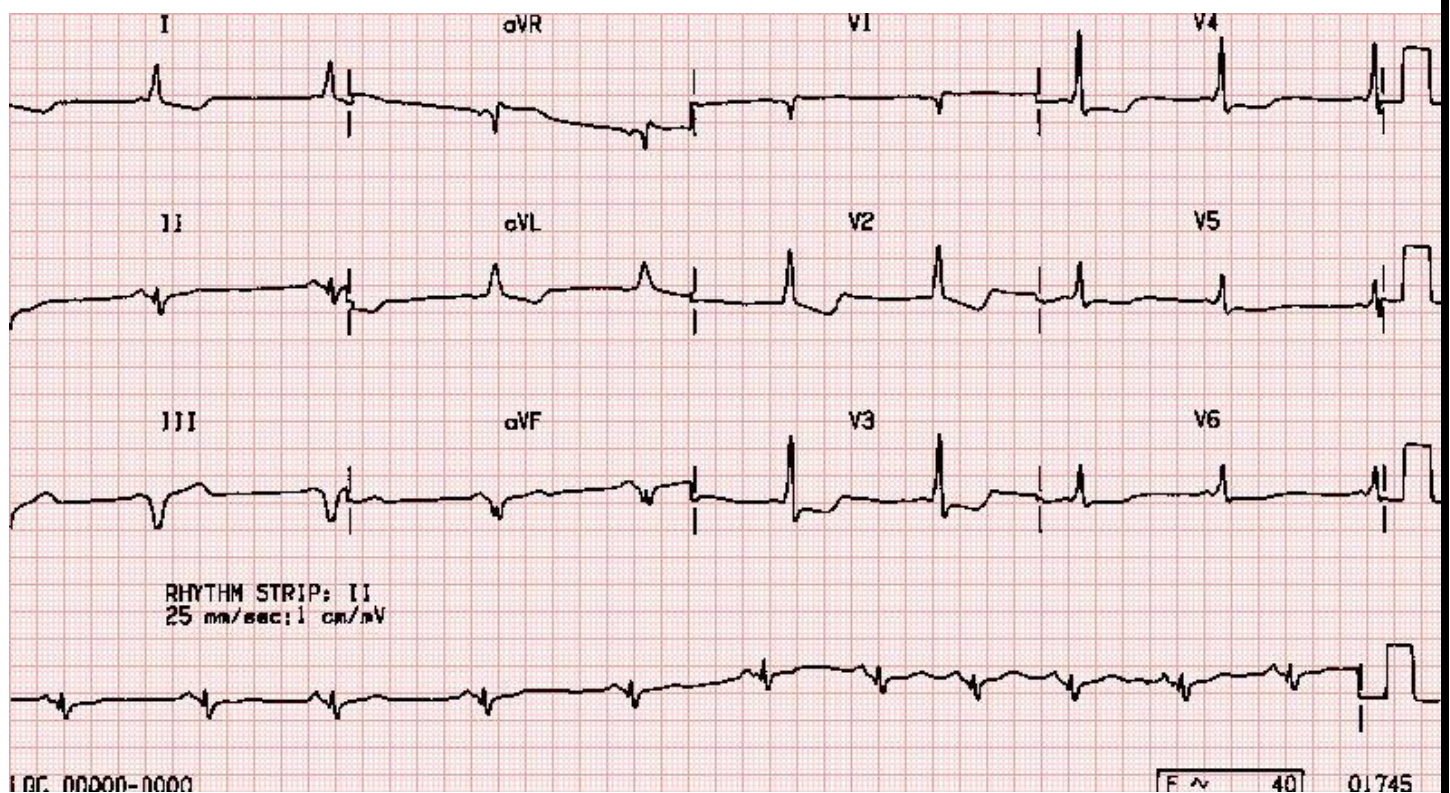
- 1) HOCM
- 2) mitral valve prolapse
- 3) Ebstein's anomaly
- 4) secundum ASD
- 5) thyrotoxicosis

Management:

- 1) Definitive treatment: **radiofrequency ablation** of the accessory pathway
- 2) Medical therapy: **sotalol****, **amiodarone**, **flecainide**

****sotalol** should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

Prominent R in V1 also in posterior wall infarction



The ECG shows a shortened PR interval (in this case the PR interval is approximately 0.08 s - two small squares NR 0.12-0.2 s) and a delta wave. In this case it is probably best seen in the lateral chest leads V3 - V6.

Ventricular pre-excitation (if there were a history of tachycardia it would be Wolff-Parkinson-White syndrome) commonly masquerades as other conditions, such as bundle branch block or ischaemia.

Atrial flutter

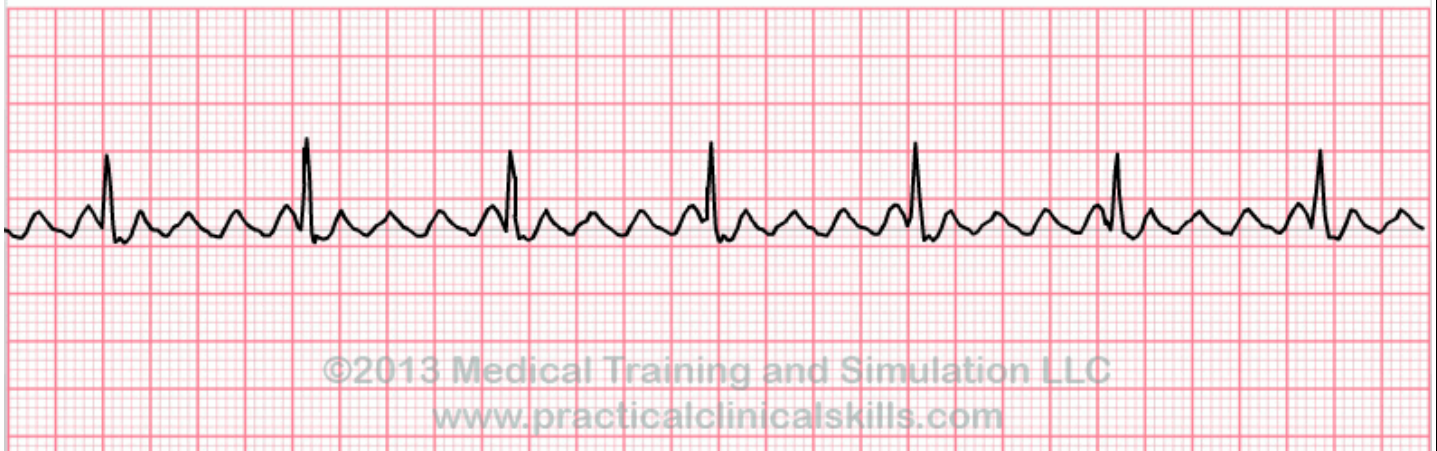
Atrial flutter is a form of supraventricular tachycardia characterised by a succession of rapid atrial depolarisation waves.

ECG findings

- 'sawtooth' appearance
- as the underlying atrial rate is often around 300/min the ventricular or heart rate is dependent on the degree of AV block. For example if there is 2:1 block the ventricular rate will be 150/min
- flutter waves may be visible following carotid sinus massage or adenosine

Management:

- 1) similar to that of atrial fibrillation although **medication may be less effective**
- 2) atrial flutter is **more sensitive to cardioversion** however so lower energy levels may be used
- 3) **radiofrequency ablation of the tricuspid valve isthmus** is curative for most patients



Atrial fibrillation

Classification:

- 1) **First detected episode** (irrespective of whether it is symptomatic or self-terminating)
- 2) **Recurrent episodes:**
When a patient has 2 or more episodes of AF:
 - ⇒ **Paroxysmal AF:**
 - ✓ If episodes of AF terminate spontaneously
 - ✓ Such episodes last less than 7 days (typically < 24 hours).
 - ⇒ **Persistent AF:**
 - ✓ If the arrhythmia is not self-terminating.
 - ✓ Such episodes usually last greater than 7 days
- 3) **Permanent AF:**
 - ⇒ Continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate
 - ⇒ Treatment goals are therefore rate control and anticoagulation if appropriate

Rate control Or maintenance of sinus rhythm

- The following is based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines

Agents used to control rate:

- 1) **beta-blockers**
- 2) **calcium channel blockers**
- 3) **digoxin**
 - ⇒ **Not considered first-line anymore** as they are less effective at controlling the rate during exercise.
 - ⇒ However, they are the preferred choice if the patient has coexistent **heart failure**

Agents used to maintain sinus rhythm:

- 1) **sotalol**
- 2) **amiodarone**
- 3) **flecainide**
- 4) others (less commonly used in UK): disopyramide, dofetilide, procainamide, propafenone, quinidine

Factors favouring rate control	Factors favouring rhythm control
• Older than 65 years • History of ischaemic heart disease	• Younger than 65 years • Symptomatic • First presentation • Lone AF or AF secondary to a corrected precipitant (e.g. Alcohol) • Congestive heart failure

Cardioversion:

Onset < 48 hours

- If the atrial fibrillation (AF) is definitely of less than 48 hours onset patients should be **heparinised**.
- Patients who have risk factors for ischaemic stroke should be put on **lifelong oral anticoagulation**.
- Otherwise, patients may be cardioverted using either:
 - 1) **electrical - 'DC cardioversion'**
 - 2) **pharmacology:**
 - ⇒ **Amiodarone** if **structural heart disease**,
 - ⇒ **Flecainide** in those **without** structural heart disease
- Following electrical cardioversion (if AF < 48 hours duration) then further anticoagulation is unnecessary

Onset > 48 hours

- 1) If the patient has been in AF for more than 48 hours then **anticoagulation** should be given for at least **3 weeks** prior to cardioversion.
- 2) **An alternative strategy** is to perform a **transoesophageal echo** (TOE) to exclude a left atrial appendage (LAA) thrombus.
 - ⇒ If excluded patients may be **heparinised** and **cardioverted** immediately.
- 3) **If there is a high risk of cardioversion failure** (e.g. Previous failure or AF recurrence) then it is recommend to have at least **4 weeks amiodarone** or **sotalol** prior to electrical cardioversion
- 4) **Following electrical cardioversion:**
 - ⇒ Patients should be **anticoagulated** for at least **4 weeks**.
 - ⇒ After this time decisions about anticoagulation should be taken on an individual basis depending on the risk of recurrence

Pharmacological cardioversion:

Agents with proven efficacy in the pharmacological cardioversion of atrial fibrillation:

- 1) Amiodarone (if structural heart disease)
- 2) flecainide (if no structural heart disease)
- 3) others (less commonly used in UK): quinidine, dofetilide, ibutilide, propafenone

Less effective agents:

- 1) beta-blockers (including sotalol)
- 2) calcium channel blockers
- 3) digoxin
- 4) disopyramide
- 5) procainamide

Anticoagulation

- The European Society of Cardiology published updated guidelines on the management of **atrial fibrillation** in **2012**.
- They suggest using the **CHA₂DS₂-VASc score** to determine the most appropriate anticoagulation strategy. This scoring system superseded the CHADS₂ score.

	Risk factor	Points
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A ₂	Age >= 75 years	2
	Age 65-74 years	1
D	Diabetes	1
S ₂	Prior Stroke or TIA	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
S	Sex (female)	1

The table below shows a suggested anticoagulation strategy based on the score:

Score	Anticoagulation
0	No treatment
1	Males: Consider anticoagulation Females: No treatment
2 or more	Offer anticoagulation

- Doctors have always thought carefully about the risk/benefit profile of starting someone on warfarin.
- A history of falls, old age, alcohol excess and a history of previous bleeding are common things that make us consider whether warfarinisation is in the best interests of the patient.
- NICE now recommend we formalise this risk assessment using the HASBLED scoring system.

	Risk factor	Points
H	Uncontrolled Hypertension, , systolic BP > 160 mmHg	1
A	Abnormal renal function (dialysis or creatinine > 200) Or Abnormal liver function (cirrhosis, bilirubin > 2 times normal, ALT/AST/ALP > 3 times normal	1 for any renal abnormalities 1 for any liver abnormalities
S	Stroke, history of	1
B	Bleeding, history of bleeding or tendency to bleed	1
L	Labile INRs (unstable/high INRs, time in therapeutic range < 60%)	1
E	Elderly (> 65 years)	1
D	Drugs Predisposing to Bleeding (Antiplatelet agents, NSAIDs) Or Alcohol Use (>8 drinks/week)	1 for drugs 1 for alcohol

Paradoxical embolisation

- For a right-sided thrombus (e.g. DVT) to cause a left-sided embolism (e.g. stroke) it must obviously pass from the right-to-left side of the heart
- **The following cardiac lesions may cause such events:**
 - ✓ **Patent foramen ovale:** present in around 20% of the population
 - ✓ **Atrial septal defect:** a much less common cause

Atrial fibrillation: post-stroke

- NICE issued guidelines on atrial fibrillation (AF) in 2006. (فديمة)
- They included advice on the management of patients with AF who develop a stroke or transient-ischaemic attack (TIA).

Recommendations include:

- 1) Following a stroke or TIA **warfarin** should be given as the anticoagulant of choice.
- 2) **Aspirin/dipyridamole** should only be given **if needed for the treatment of other comorbidities** (I think there in new guidelines plavix should be given in case of stroke but not TIA)
- 3) In acute stroke patients, in the **absence of haemorrhage**, anticoagulation therapy should be commenced after **2 weeks**.
- 4) If imaging shows a **very large cerebral infarction** then the initiation of **anticoagulation should be delayed**

Long QT syndrome

- An **inherited** condition associated with **delayed repolarization of the ventricles**.
- It is important to recognise as it may lead to **ventricular tachycardia** and can therefore cause **collapse/sudden death**.
- The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier **potassium channel**.
- A normal corrected QT interval is less than **430 ms in males** and **450 ms in females**.

Causes of a prolonged QT interval:

Congenital	Drugs*	Other
<u>Jervell-Lange-Nielsen syndrome</u> (includes deafness and is due to an abnormal potassium channel)	1) amiodarone, sotalol, 2) class 1a antiarrhythmic 3) tricyclic antidepressants, 4) SSRI (especially citalopram) 5) haloperidol 6) methadone 7) chloroquine 8) terfenadine** 9) erythromycin	1) electrolyte: ⇒ hypocalcaemia, ⇒ hypokalaemia, ⇒ hypomagnesaemia 2) hypothermia 3) subarachnoid haemorrhage 4) acute myocardial infarction 5) myocarditis
<u>Romano-Ward syndrome</u> (no deafness)		

Features:

- 1) may be picked up on routine ECG or following family screening
- 2) sudden cardiac death
- 3) **Long QT1:**
⇒ Usually associated with exertional syncope, often **swimming**
- 4) **Long QT2:**
⇒ Often associated with syncope occurring following emotional stress, **exercise** or **auditory stimuli**
- 5) **Long QT3:** events often occur at night or **at rest**

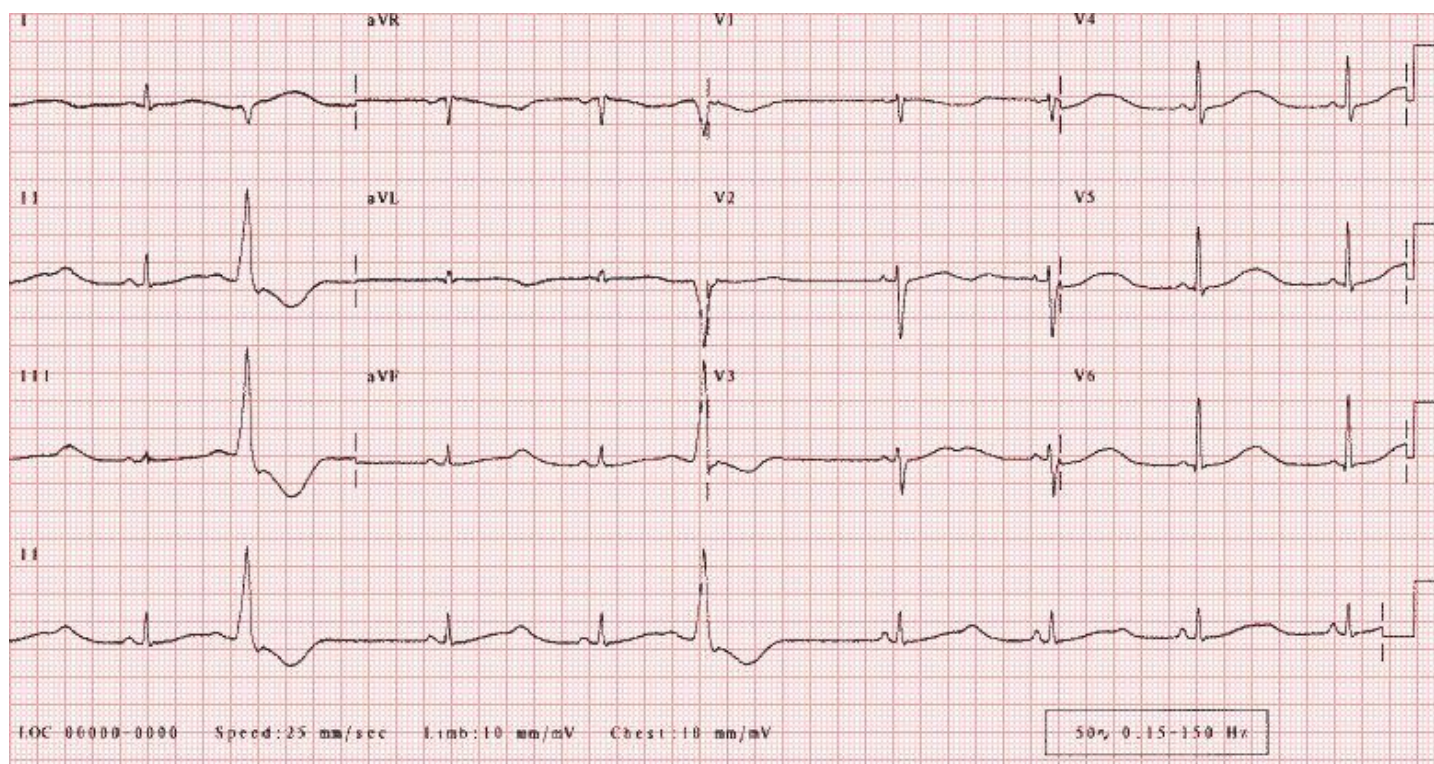
Management:

- 1) **avoid drugs which prolong the QT interval** and other precipitants if appropriate (e.g. Strenuous exercise)
- 2) **beta-blockers**
- 3) **ICD** in high risk cases.

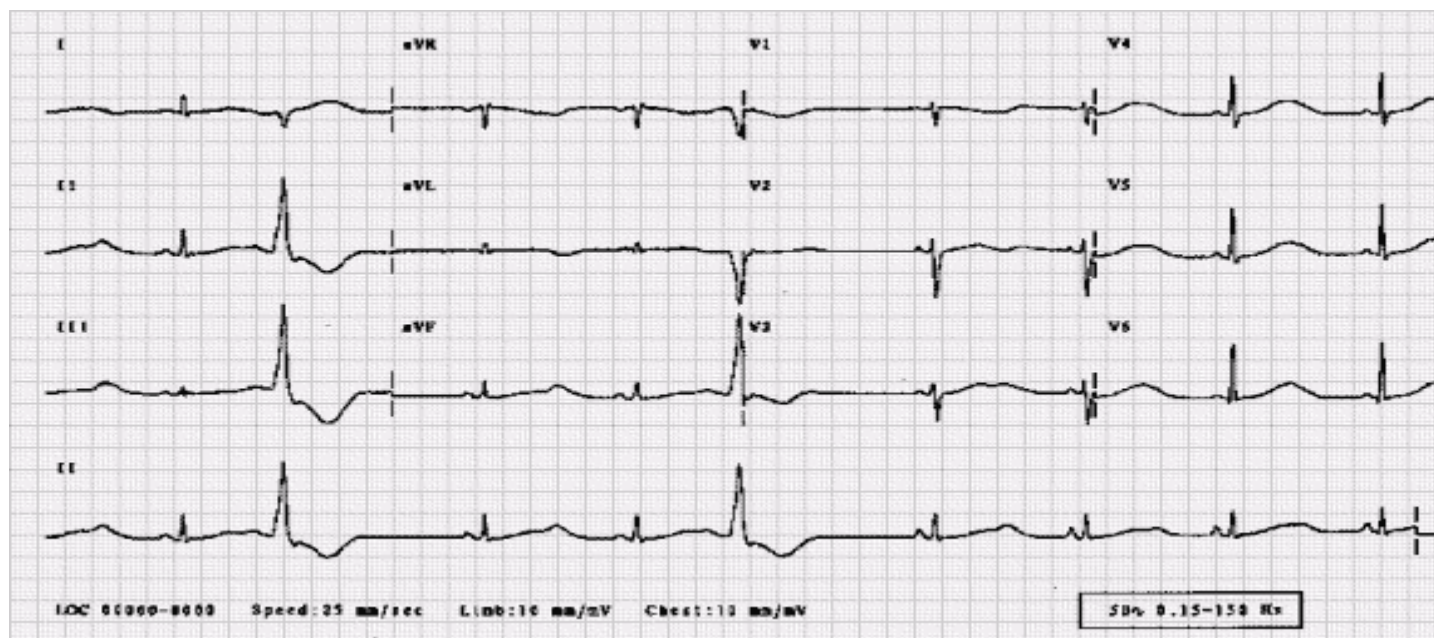
(Avoid sotalol may exacerbate long QT syndrome)

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels.

**a non-sedating antihistamine are classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time



The ECG shows a long QT and ventricular premature beats



Long QT interval

Broad complex tachycardia

- Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that **in a peri-arrest situation it is assumed to be ventricular in origin**
- **Features suggesting VT rather than SVT with aberrant conduction:**
 - 1) AV dissociation
 - 2) fusion or capture beats
 - 3) positive QRS concordance in chest leads
 - 4) marked left axis deviation
 - 5) history of IHD
 - 6) lack of response to adenosine or carotid sinus massage
 - 7) QRS > 160 ms

Ventricular tachycardia

- VT is broad-complex tachycardia originating from a **ventricular ectopic focus**.
- It has the potential to precipitate **ventricular fibrillation** and hence **requires urgent treatment**.

There are two main types of VT:

- 1) **Monomorphic VT:** most commonly caused by **myocardial infarction**
- 2) **Polymorphic VT:** A subtype of polymorphic VT is **torsades de pointes** which is precipitated by **prolongation of the QT interval**.

Management:

- 1) **If the patient has adverse signs** (systolic BP < 90 mmHg, chest pain, heart failure OR rate > 150 beats/min) then **immediate cardioversion** is indicated.
- 2) **In the absence of such signs antiarrhythmics** may be used. If these fail, **then electrical cardioversion** may be needed with **synchronised DC shocks**

Drug therapy:

- 1) **amiodarone:** ideally administered through a central line
- 2) **lidocaine:** use with caution in severe left ventricular impairment
- 3) **procainamide**

Verapamil should NOT be used in VT

If drug therapy fails:

- 1) **electrophysiological study (EPS)**
- 2) **implantable cardioverter-defibrillator (ICD)** - this is particularly indicated in patients with significantly impaired LV function



There is a broad complex rapid tachycardia typical of VT starting halfway through this ECG. The first half of the recording gives a clue to the aetiology of the VT. There is T wave inversion and slight ST elevation in the inferior leads suggesting the evolving changes of an acute inferior myocardial infarction. **Monomorphic VT**

Torsades de pointes

- Torsades de pointes ('twisting of the points') is a rare arrhythmia associated with a **long QT interval**.
- It may deteriorate into **ventricular fibrillation** and hence lead to **sudden death**

Causes of long QT interval:

- 1) **congenital:** Jervell-Lange-Nielsen syndrome, Romano-Ward syndrome
- 2) **antiarrhythmics:** amiodarone, sotalol, class 1a antiarrhythmic drugs
- 3) tricyclic antidepressants
- 4) antipsychotics
- 5) chloroquine
- 6) terfenadine
- 7) erythromycin
- 8) **electrolyte:** hypocalcaemia, hypokalaemia, hypomagnesaemia
- 9) myocarditis
- 10) hypothermia
- 11) subarachnoid haemorrhage

Management:

- **IV magnesium sulphate** 2 g IV over 10 minutes

Antiarrhythmics

Vaughan Williams classification

- The Vaughan Williams classification of antiarrhythmics is still widely used although it should be noted that a number of common drugs are not included in the classification e.g. adenosine, atropine, digoxin and magnesium

AP = action potential

Class	Examples	Mechanism of action	Notes
Ia	1) Quinidine 2) Procainamide 3) Disopyramide	1) Block sodium channels 2) Increases AP duration	- Quinidine toxicity causes cinchonism (headache, tinnitus, thrombocytopaenia) - Procainamide may cause drug-induced lupus
Ib	1) Lidocaine 2) Mexiletine 3) Tocainide	1) Block sodium channels 2) Decreases AP duration	
Ic	1) Flecainide 2) Encainide 3) Propafenone	1) Block sodium channels 2) No effect on AP duration	
II	1) Propranolol 2) Atenolol 3) Bisoprolol 4) Metoprolol	Beta-adrenoceptor antagonists	
III	1) Amiodarone 2) Sotalol 3) Ibutilide 4) Bretylium	Block potassium channels	
IV	1) Verapamil 2) Diltiazem	Calcium channel blockers	

- ☒ **Adenosine is safe in pregnancy.**
- ☒ Some beta blockers can be used in pregnancy - there is small risk of fetal bradycardia and intrauterine growth retardation.
- ☒ Verapamil should not be used in the first trimester.
- ☒ Amiodarone should be avoided unless no alternative.

Flecainide

- Flecainide is a Vaughan Williams **class 1c** antiarrhythmic.
- It slows conduction of the action potential by acting as a potent **sodium channel blocker**.
- This may be reflected by **widening of the QRS** complex and **prolongation of the PR interval**
- The Cardiac Arrhythmia Suppression Trial (CAST, 1989) investigated the use of agents to treat asymptomatic or mildly symptomatic premature ventricular complexes (PVCs) post myocardial infarction. The hypothesis was that this would reduce deaths from ventricular arrhythmias. **Flecainide was actually shown to increase mortality post myocardial infarction and is therefore contraindicated in this situation**

Indications:

- 1) atrial fibrillation
- 2) SVT associated with accessory pathway e.g. Wolf-Parkinson-White syndrome

Adverse effects:

- 1) negatively inotropic
- 2) bradycardia
- 3) proarrhythmic
- 4) oral paraesthesia
- 5) visual disturbances

Amiodarone

- **Class III** antiarrhythmic agent used in the treatment of **atrial**, **nodal** and **ventricular** tachycardias.
- The main mechanism of action is by **blocking potassium channels** which inhibits repolarisation and hence **prolongs the action potential**.
- Amiodarone also has other actions such as **blocking sodium channels** (a class I effect)

The use of amiodarone is limited by a number of factors:

- 1) long half-life (20-100 days)
- 2) should ideally be given into central veins (causes thrombophlebitis)
- 3) has proarrhythmic effects due to lengthening of the QT interval
- 4) interacts with drugs commonly used concurrently e.g. **Decreases metabolism of warfarin**
- 5) numerous long-term adverse effects (see below)

Amiodarone adverse effects:

- 1) **thyroid dysfunction**: both hypothyroidism and hyperthyroidism
- 2) **corneal deposits**: present in most patients, rarely interfere with vision, usually reversible on withdrawal of drug
- 3) photosensitivity
- 4) pulmonary fibrosis/pneumonitis
- 5) liver cirrhosis/hepatitis
- 6) peripheral neuropathy, myopathy
- 7) **'slate-grey' appearance**
- 8) prolonged QT interval
- 9) thrombophlebitis and injection site reactions
- 10) bradycardia

Important drug interactions of amiodarone include:

- 1) decreased metabolism of warfarin, therefore increased INR
- 2) increased digoxin levels

Monitoring of patients taking amiodarone

- TFT, LFT, U&E, CXR prior to treatment
- TFT, LFT every 6 months

Amiodarone and the thyroid gland

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

A) Amiodarone-induced hypothyroidism (AIH)

- The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to **the high iodine content** of amiodarone causing a **Wolff-Chaikoff effect***
- Amiodarone **may be continued** if this is desirable

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

B) Amiodarone-induced thyrotoxicosis (AIT)

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	AIT type 2
Pathophysiology	Excess iodine-induced thyroid hormone synthesis Jod-Basedow effect	Amiodarone-related destructive thyroiditis
radioiodine uptake scan	High uptake	low uptake
Goiter	Goitre Present	Absent (tender thyroid)
Management	Carbimazole or potassium perchlorate	Corticosteroids

- Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT
- In some patients it may not be possible to stop the amiodarone but there must be a strong clinical indication for this **such as VT/VF**.
- **Colour flow Doppler** has been investigated as a tool to differentiate type 1 and type 2 AIT in a number of studies
- **Amiodarone:**
 - ⇒ Inhibits the peripheral conversion of thyroxine (T4) to tri-iodothyronine (T3) so consequently T4 may be elevated and T3 low.
 - ⇒ As T3 is the most active thyroid hormone the low T3 feedbacks at the pituitary level result in increased TSH secretion.
- **Lithium** cause hypothyroid; low T4 and elevated thyroid-stimulating hormone (TSH).

Adenosine

Mechanism of action:

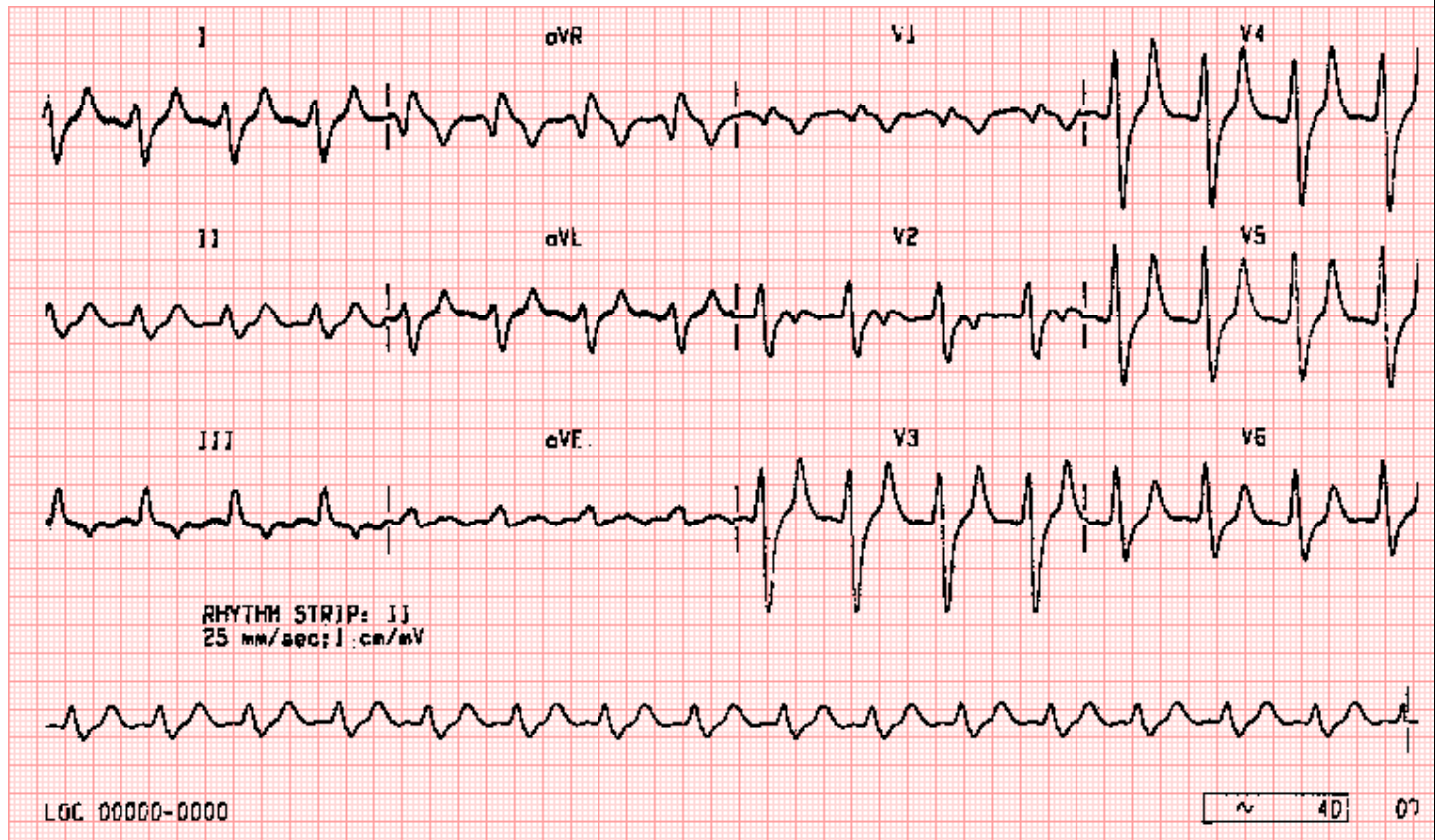
- causes **transient heart block in the AV node**
- agonist of the A1 receptor which **inhibits adenylyl cyclase** thus **reducing cAMP** and causing **hyperpolarization** by **increasing outward potassium flux**
- adenosine has a very short half-life of about **8-10 seconds**

The effects of adenosine are:

- ⇒ Enhanced by dipyridamole (anti-platelet agent) and
- ⇒ blocked by theophyllines
- It should be avoided in asthmatics due to possible bronchospasm.

Adverse effects:

- 1) chest pain
- 2) bronchospasm
- 3) can enhance conduction down accessory pathways, resulting in increased ventricular rate (e.g. WPW syndrome)



The ECG shows quite tall and pointed T waves. They 'would hurt if you sat on them'. These are the so-called 'tented T-waves' of **hyperkalaemia** and are quite distinctive. Also there is widening of the QRS and a reduction in the size of the P wave which is barely visible.

ECG: axis deviation

Causes of left axis deviation (LAD)

- 1) left anterior hemiblock
- 2) left bundle branch block
- 3) Wolff-Parkinson-White syndrome* - right-sided accessory pathway
- 4) hyperkalaemia
- 5) congenital: ostium primum ASD, tricuspid atresia
- 6) minor LAD in obese people

*in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation

Causes of right axis deviation (RAD)

- 1) right ventricular hypertrophy
- 2) chronic lung disease → cor pulmonale
- 3) pulmonary embolism
- 4) left posterior hemiblock
- 5) ostium secundum ASD
- 6) Wolff-Parkinson-White syndrome* - left-sided accessory pathway
- 7) normal in infant < 1 years old
- 8) minor RAD in tall people

ECG: hypothermia

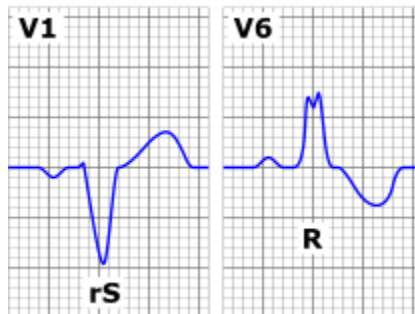
The following ECG changes may be seen in hypothermia

- bradycardia
- 'J' wave - small hump at the end of the QRS complex
- first degree heart block
- long QT interval
- atrial and ventricular arrhythmias

Left ventricular hypertrophy (LVH), that is, sum of the S wave in V1 and R wave in V6 is >35 mm.

ECG: left bundle branch block

The diagram below shows the typical features of left bundle branch block (**LBBB**):



One of the most common ways to remember the difference between LBBB and RBBB is **WiLLiaM MaRRoW**

- In **LBBB** there is a 'W' in V1 and a 'M' in V6
- In **RBBB** there is a 'M' in V1 and a 'W' in V6



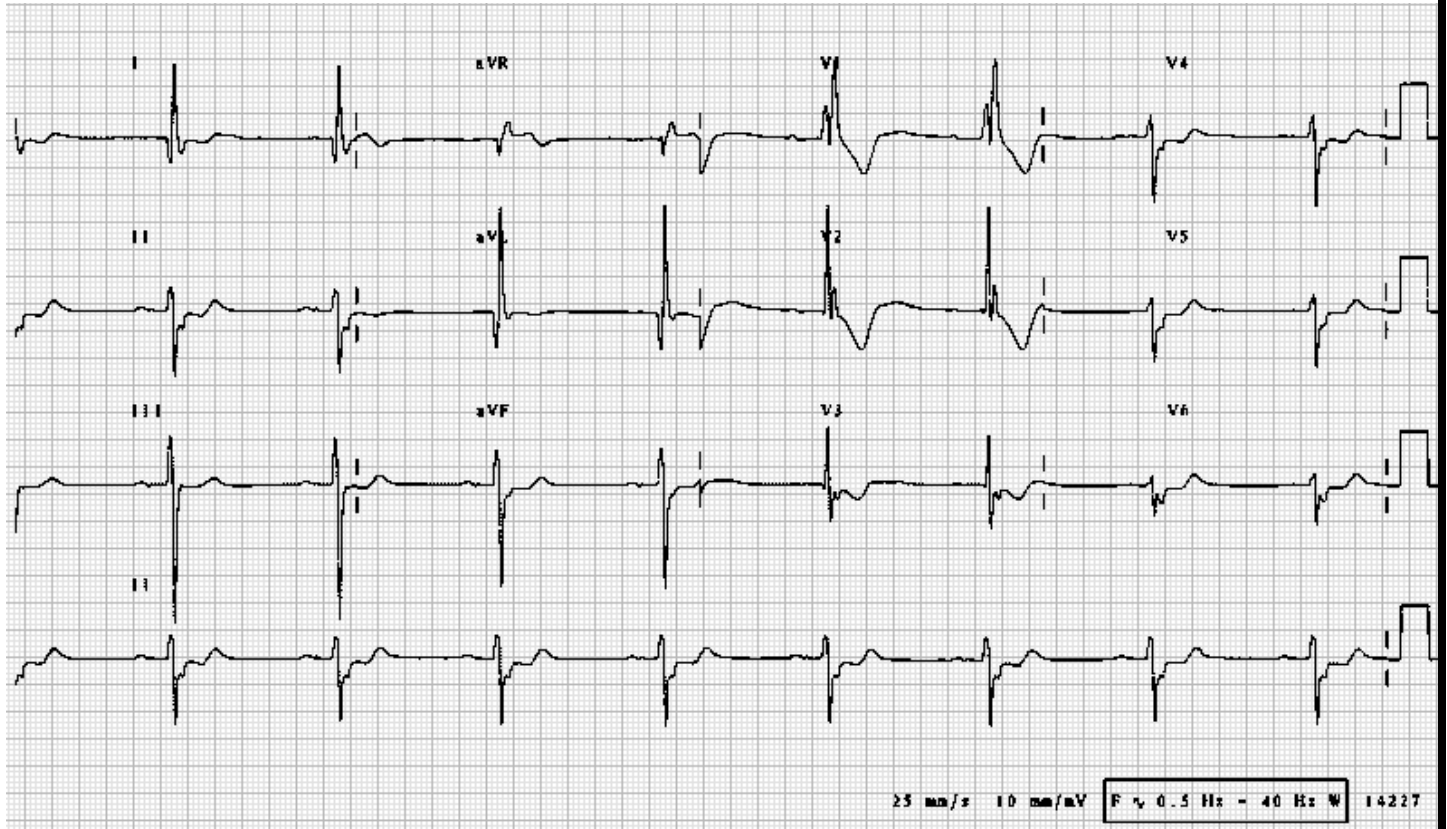
ECG showing typical features of LBBB

Causes of LBBB:

- ischaemic heart disease
- hypertension
- aortic stenosis
- cardiomyopathy
- rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

'trifascicular block' which is not strictly an ECG diagnosis but is a term used for the combination of **right bundle branch block**, **left hemiblock** (typically left anterior hemiblock) and **long PR interval**.

It implies that the bundle branches (Purkinje fibres) are blocked in the right bundle and one of the left hemibundles. The 'third' bundle is also delayed or partially blocked hence the name. However, the delay (long PR interval) is usually at the AV node.



Heart block

First degree heart block:

⇒ PR interval > 0.2 seconds



Second degree heart block:

Type 1 (Mobitz I, Wenckebach):

⇒ progressive prolongation of the PR interval until a dropped beat occurs

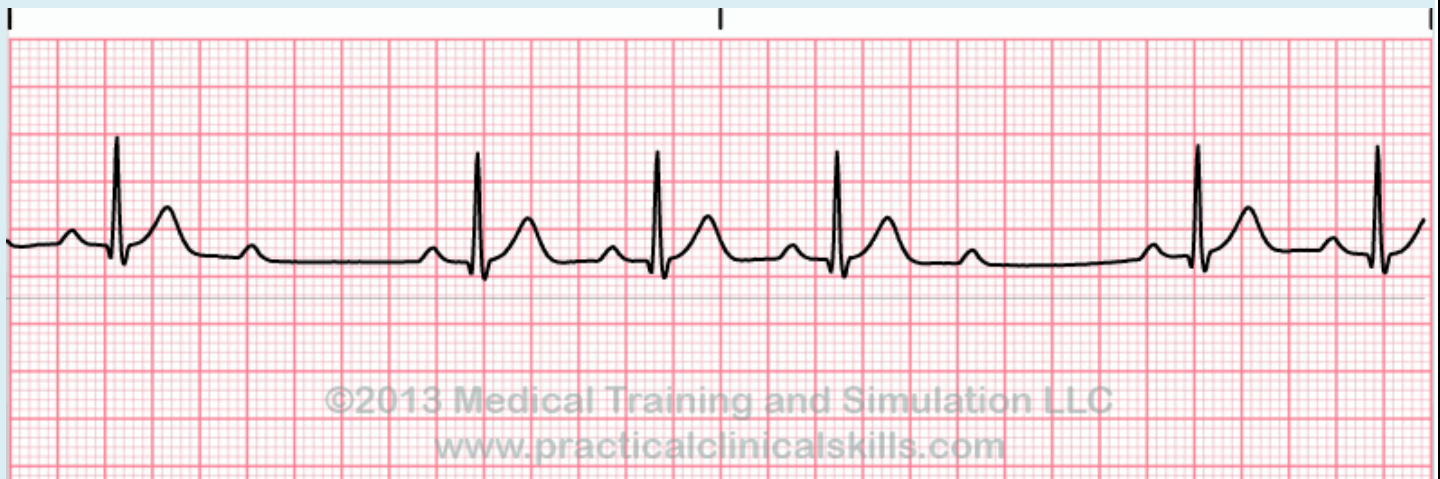


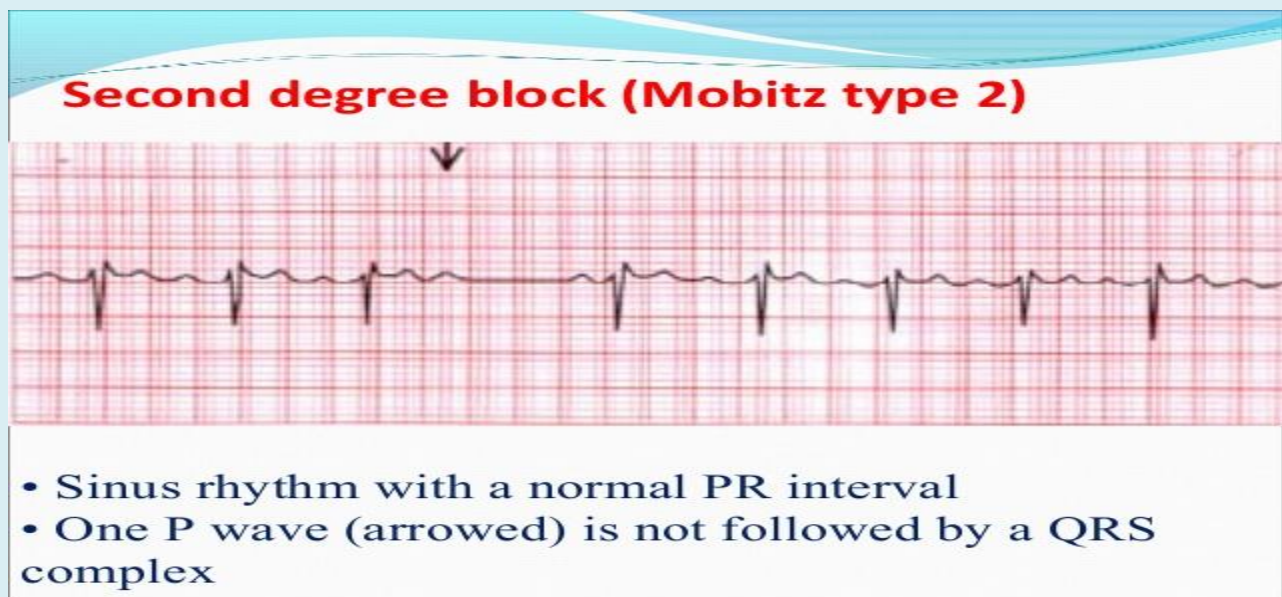
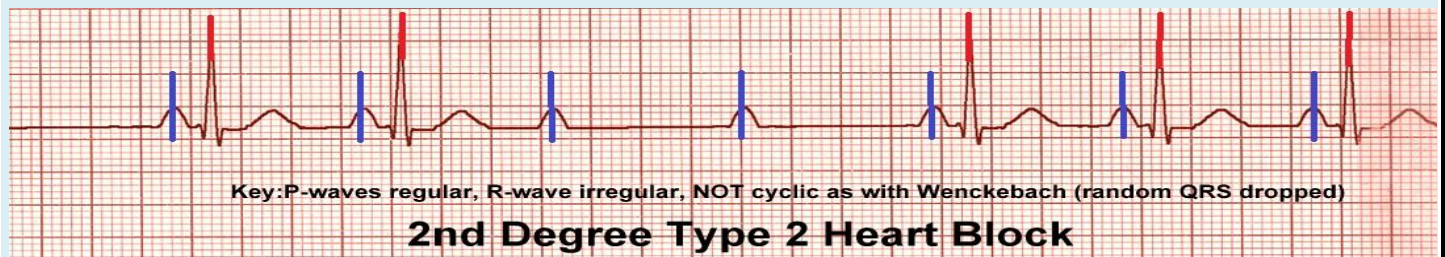
Mobitz Type I (Wenckebach) Second-Degree AV Block



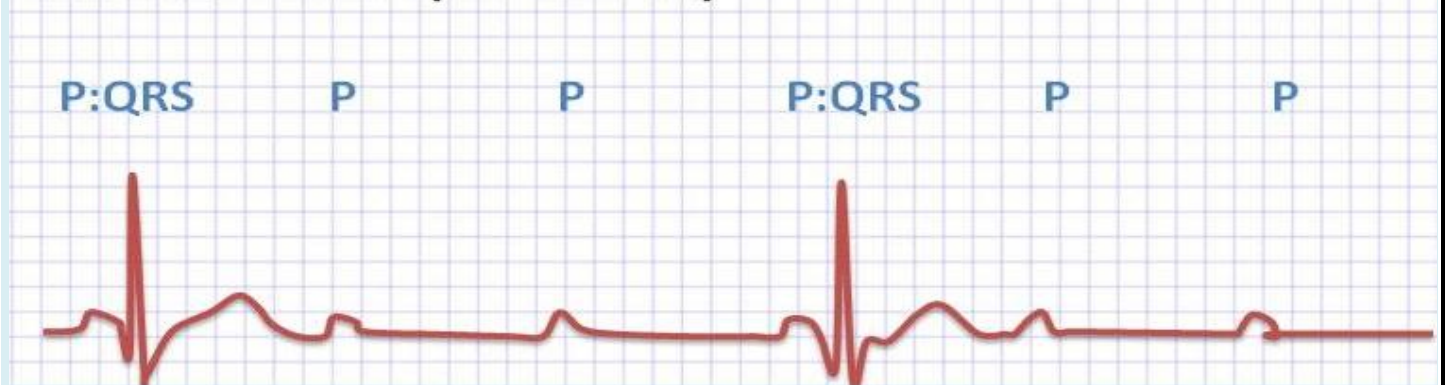
Type 2 (Mobitz II):

⇒ PR interval is constant but the P wave is often not followed by a QRS complex

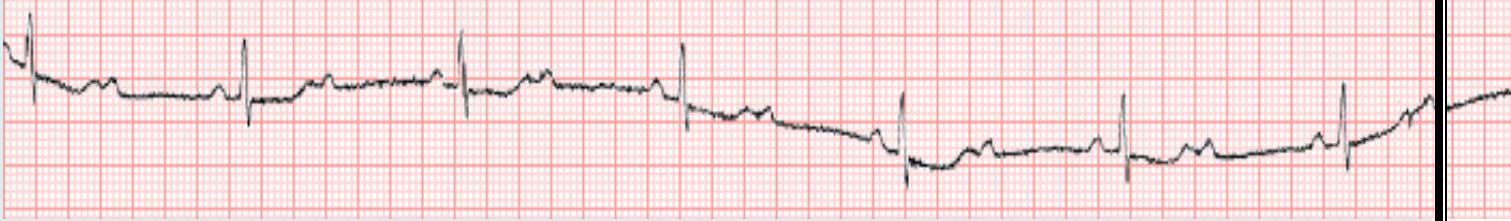




MOBITZ TYPE 2 (3:1 BLOCK)



II



There are two P waves for each QRS.

The conducted PR intervals are constant.

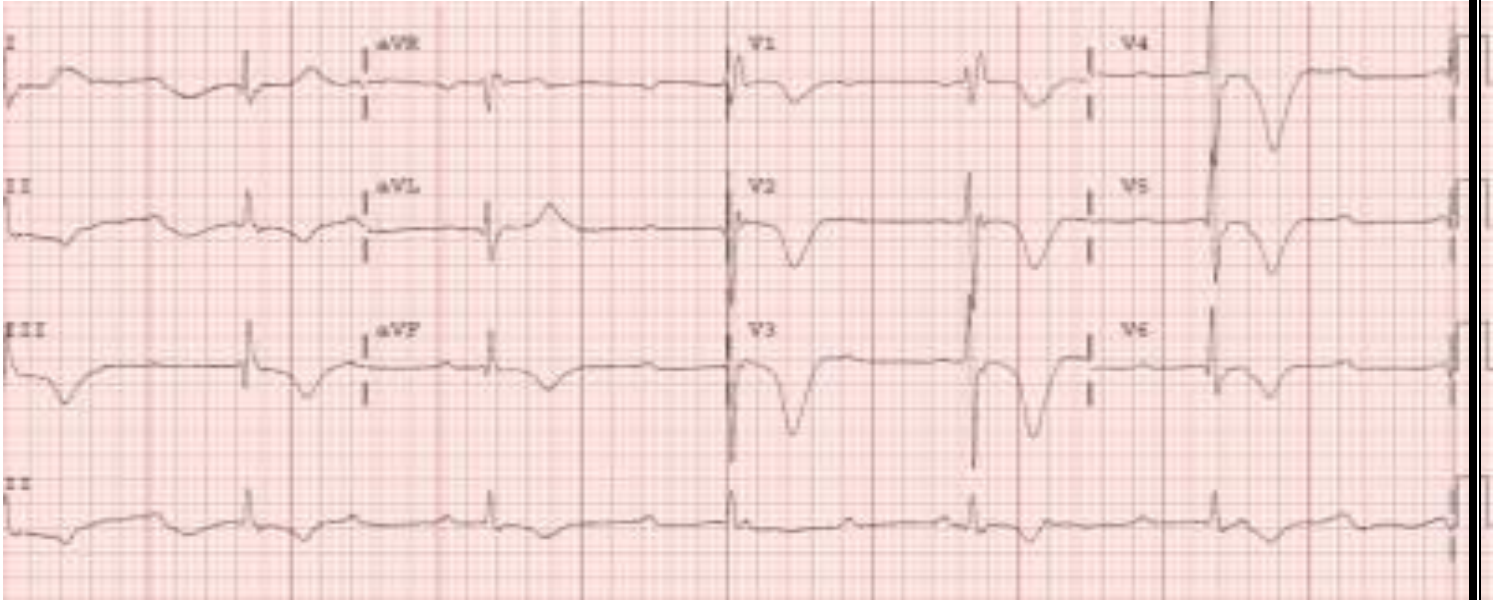
2:1 AV block cannot be classified as Mobitz type 1 or type 2 AV block since it would not be known whether the second P wave would be conducted with the same or a prolonged PR interval.

Third degree (complete) heart block

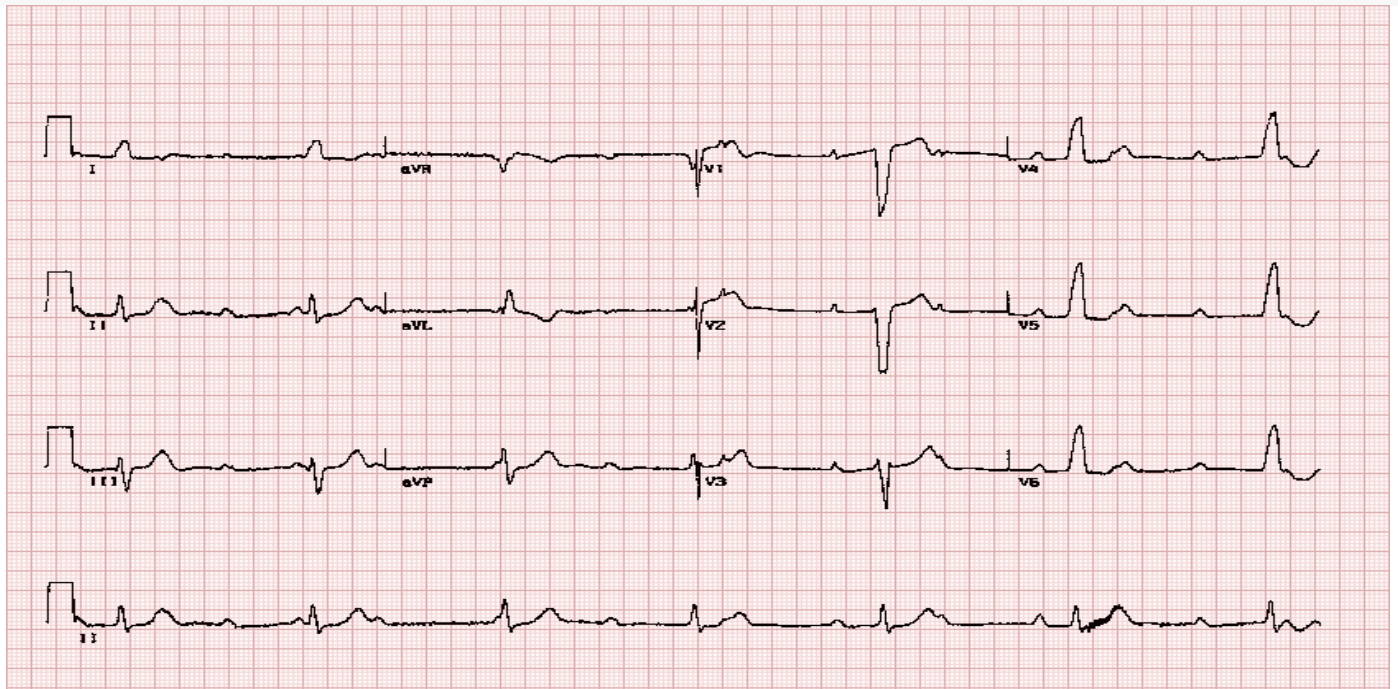
⇒ there is no association between the P waves and QRS complexes

Features:

- 1) syncope
- 2) heart failure
- 3) regular bradycardia (30-50 bpm)
- 4) wide pulse pressure
- 5) JVP: cannon waves in neck
- 6) variable intensity of S1



ECG showing third degree (complete) heart block



The ECG shows complete heart block. There are P waves which show no relation to the QRS complexes. The QRS complexes are wide, regular and represent a ventricular escape rhythm.

Peri-arrest rhythms

Bradycardia

The 2010 Resuscitation Council UK guidelines: the management of bradycardia depends on:

- 1) **Identifying the presence of adverse signs** (signs indicating haemodynamic compromise)
- 2) **Identifying the potential risk of asystole**

Adverse signs:

The following factors indicate haemodynamic compromise and hence the need for treatment:

- 1) **shock:**
 - ⇒ hypotension (systolic blood pressure < 90 mmHg),
 - ⇒ pallor, sweating, cold, clammy extremities,
 - ⇒ confusion or impaired consciousness

- 2) **syncope**
- 3) **myocardial ischaemia**
- 4) **heart failure**

Management:

- 1) **Atropine** is **the first line treatment** in this situation.
- 2) **If this fails to work**, or there is the potential **risk of asystole** then **transvenous pacing** is indicated

Potential risk of asystole:

The following indicate a potential risk of asystole and hence the need for treatment with **transvenous pacing**:

- 1) complete heart block with broad complex QRS
- 2) Mobitz type II AV block
- 3) recent asystole
- 4) ventricular pause > 3 seconds

If there is a delay in the provision of transvenous pacing the following interventions may be used:

- 1) **Atropine**, up to maximum of **3mg**
- 2) **Transcutaneous pacing**
- 3) **Adrenaline** infusion titrated to response

Mobitz type II or complete heart block does not respond to atropine. Atropine may be useful for sinus or junctional bradycardia.

- Cardiac transplant pt the pharmacotherapy of bradycardias is different for this group of patients. This is because the transplanted heart is denervated.
- Therefore there is no place for atropine, even at higher doses,
- **Glucagon** can be useful if there is a suggestion of betablocker overdose
- The Resuscitation Council (UK) guidelines suggest using theophylline as a slow intravenous infusion (100-200 mg).

Peri-arrest tachycardia

- The 2010 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias.
- Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm
- Following basic ABC assessment, **patients are classified as being stable or unstable** according to the presence of any **adverse signs**
- **If any of the above adverse signs** are present then **synchronised DC shocks** should be given (under GA or conscious sedation)
- **Treatment following this is given according to whether the QRS complex is narrow or broad** and whether the rhythm is **regular or irregular**.
- The full treatment algorithm can be found at the Resuscitation Council website, below is a very limited summary:

1) Broad-complex tachycardia

Regular:

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of **amiodarone** followed by 24 hour infusion

Irregular:

1. **Polymorphic VT** (e.g. Torsade de pointes) - **IV magnesium**
2. **AF with bundle branch block** - treat as for **narrow complex tachycardia**.

2) Narrow-complex tachycardia

Regular: SVT نعتبرها

- **Vagal manoeuvres** followed by **IV adenosine**
- If above unsuccessful consider diagnosis of **atrial flutter** and control rate (e.g. **Beta-blockers**)

Irregular: AF نعتبرها

- probable **atrial fibrillation**
- **If onset < 48 hr** consider electrical or chemical cardioversion
- **If onset > 48 hr** then rate control (e.g. Beta-blocker or digoxin) and anticoagulation

Pacemakers

Temporary:

Indications for a temporary pacemaker

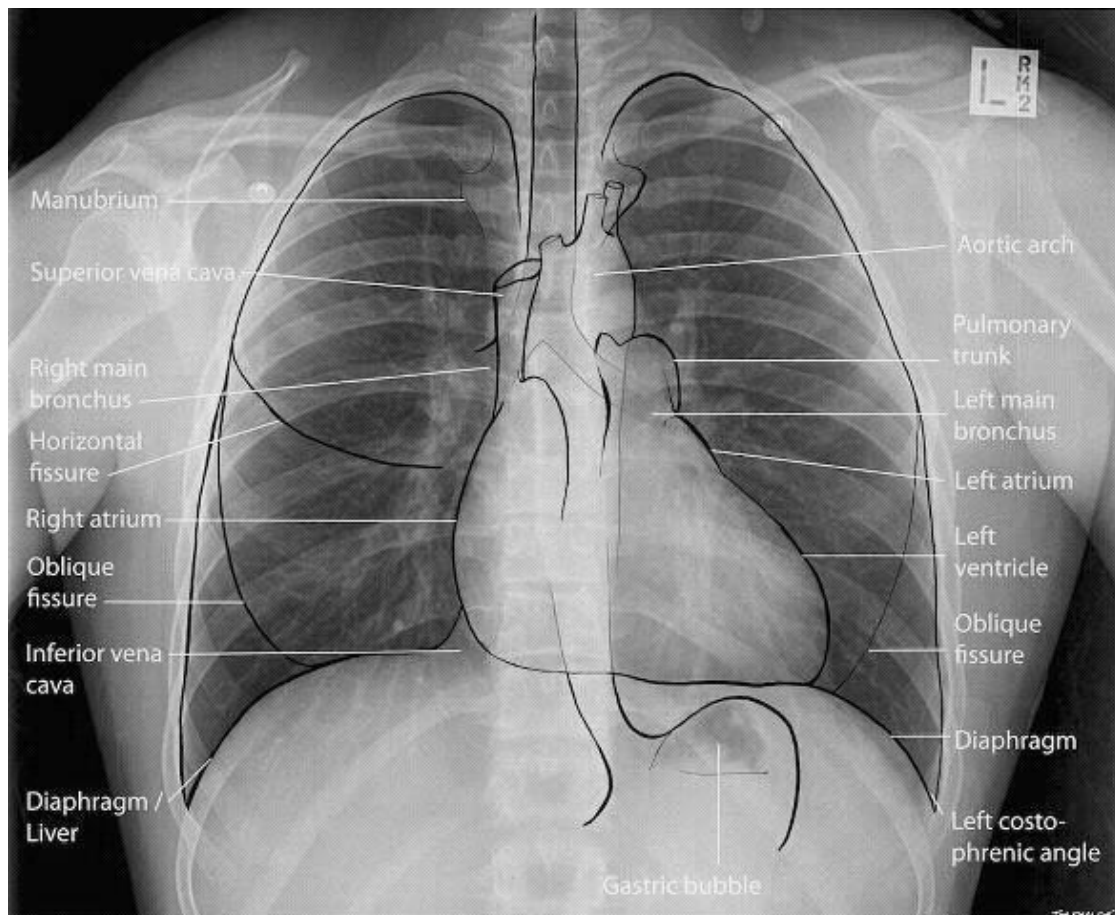
- 1) Symptomatic/haemodynamically unstable **bradycardia**, not responding to atropine
- 2) **Post-Anterior MI**: type 2 or complete **heart block***
- 3) **Trifascicular block** prior to surgery

*Post-INFERIOR MI complete heart block is common and can be managed conservatively if asymptomatic and haemodynamically stable

Implantable cardiac defibrillators:

Indications

- 1) long QT syndrome
- 2) hypertrophic obstructive cardiomyopathy
- 3) Brugada syndrome
- 4) previous cardiac arrest due to VT/VF
- 5) previous myocardial infarction with non-sustained VT on 24 hr monitoring,
- 6) inducible VT on electrophysiology testing and ejection fraction < 35%



Coarctation of the aorta

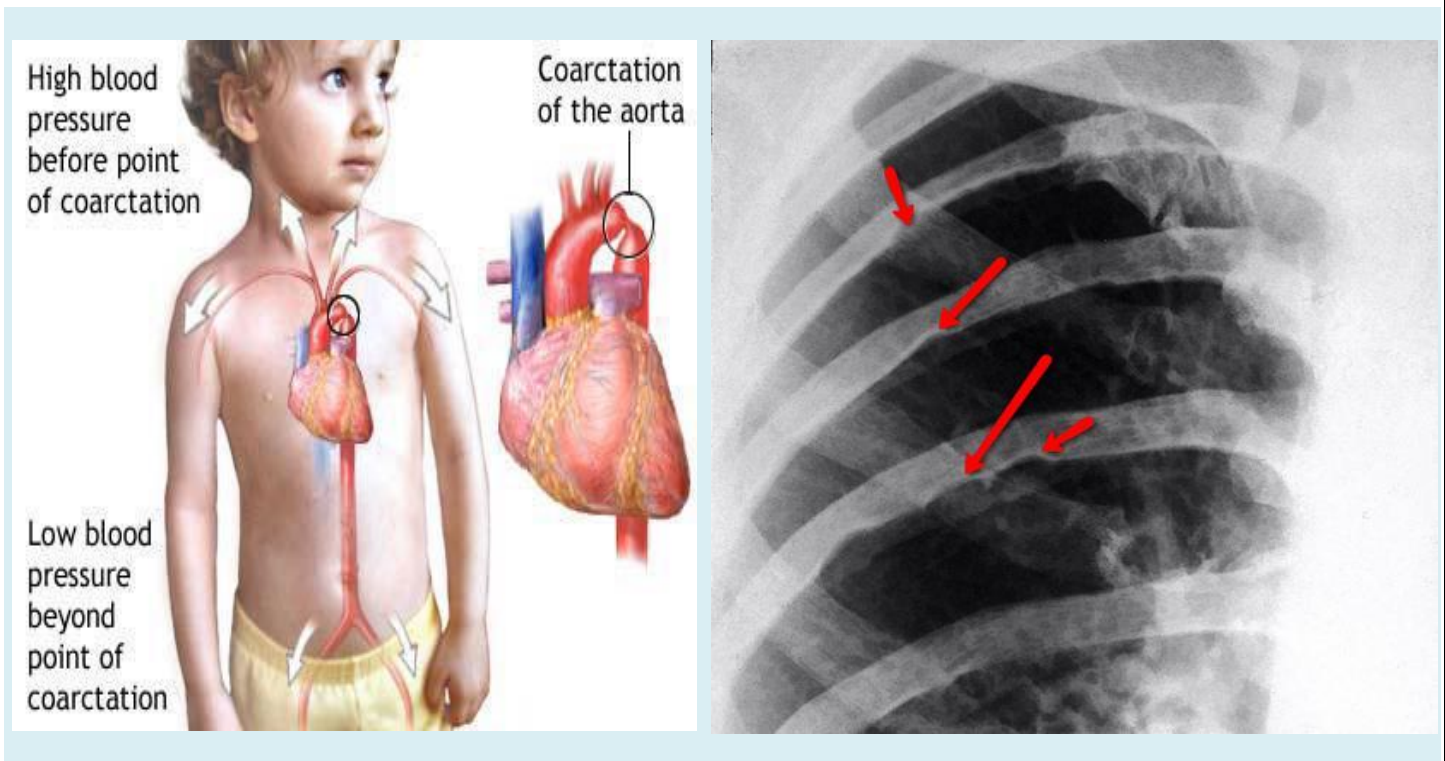
- Coarctation of the aorta describes a **congenital narrowing of the descending aorta**.
- more common in **males** (despite association with Turner's syndrome)

Features:

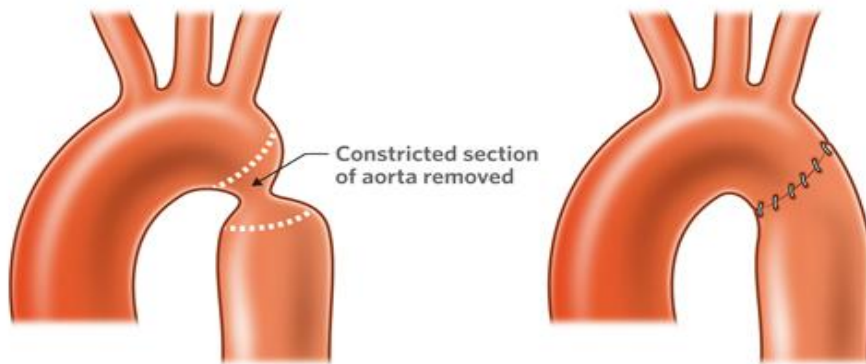
- 1) infancy: heart failure
- 2) adult: hypertension
- 3) radio-femoral delay
- 4) mid systolic murmur, **maximal over back**
- 5) apical click from the aortic valve
- 6) CXR: notching of the inferior border of the ribs (due to collateral vessels) is not seen in young children

Associations:

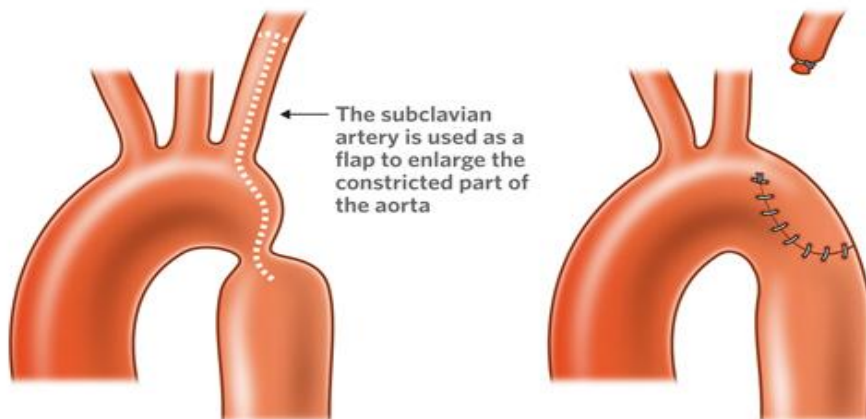
- 1) Turner's syndrome
- 2) bicuspid aortic valve
- 3) berry aneurysms
- 4) neurofibromatosis



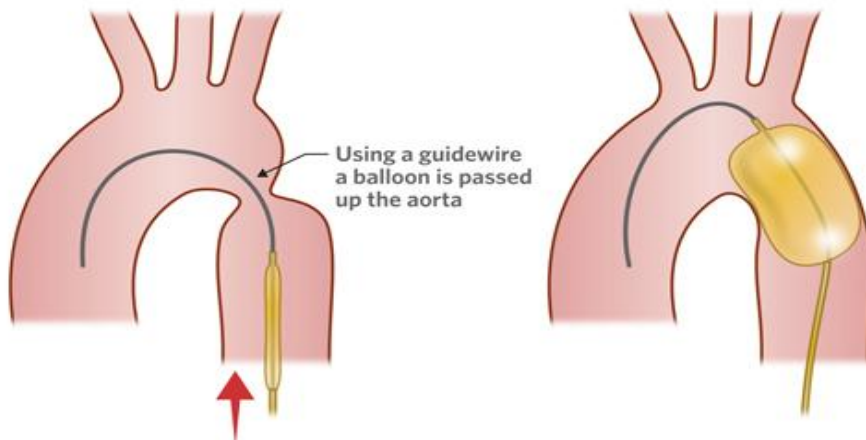
Coarctation repair



Resection and end-to-end anastomosis



Subclavian flap



Balloon angioplasty

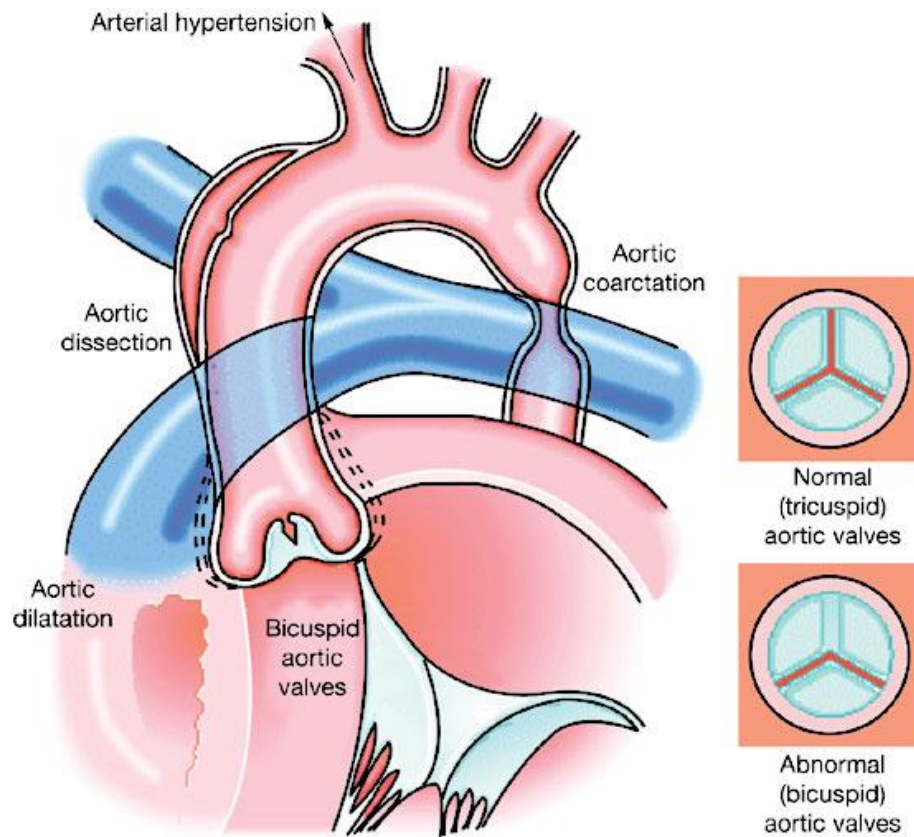


Illustration showing the typical congenital cardiovascular malformations seen in Turner syndrome: coarctation of the aorta, and bicuspid aortic valves, as well as the occurrence of aortic dilatation and dissection

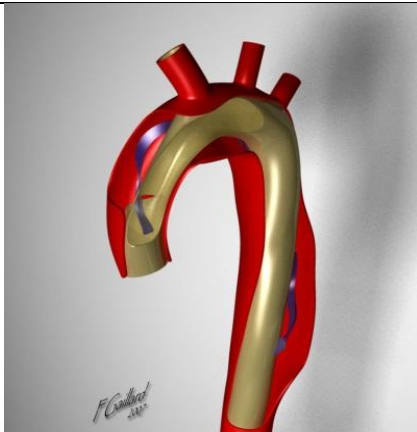
Aortic dissection

Stanford classification:

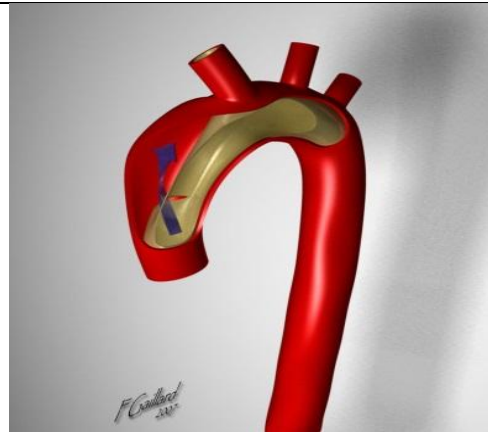
- 1) **Type A:** ascending aorta, 2/3 of cases
- 2) **Type B:** descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification:

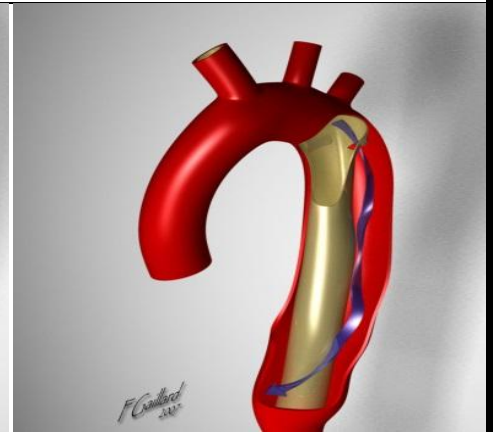
- 1) **Type I:** originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- 2) **Type II:** originates in and is confined to the ascending aorta
- 3) **Type III:** originates in descending aorta, rarely extends proximally but will extend distally



Stanford type A / DeBakey type I



Stanford type A / DeBakey type II



Stanford type B / DeBakey type III

Associations:

- 1) hypertension
- 2) trauma
- 3) bicuspid aortic valve
- 4) collagens: Marfan's syndrome, Ehlers-Danlos syndrome
- 5) Turner's and Noonan's syndrome
- 6) pregnancy
- 7) syphilis

Complications of backward tear:

- 1) aortic incompetence/regurgitation
- 2) MI: inferior pattern often seen due to right coronary involvement

Complications of forward tear

- 1) unequal arm pulses and BP
- 2) stroke
- 3) renal failure

Management:

Type A:

- **surgical management**, but blood pressure should be controlled to a **target systolic of 100-120 mmHg** whilst awaiting intervention

Type B:

- 1) conservative management
- 2) bed rest
- 3) reduce blood pressure IV **labetalol** to prevent progression

*endovascular repair of type B aortic dissection may have a role in the future

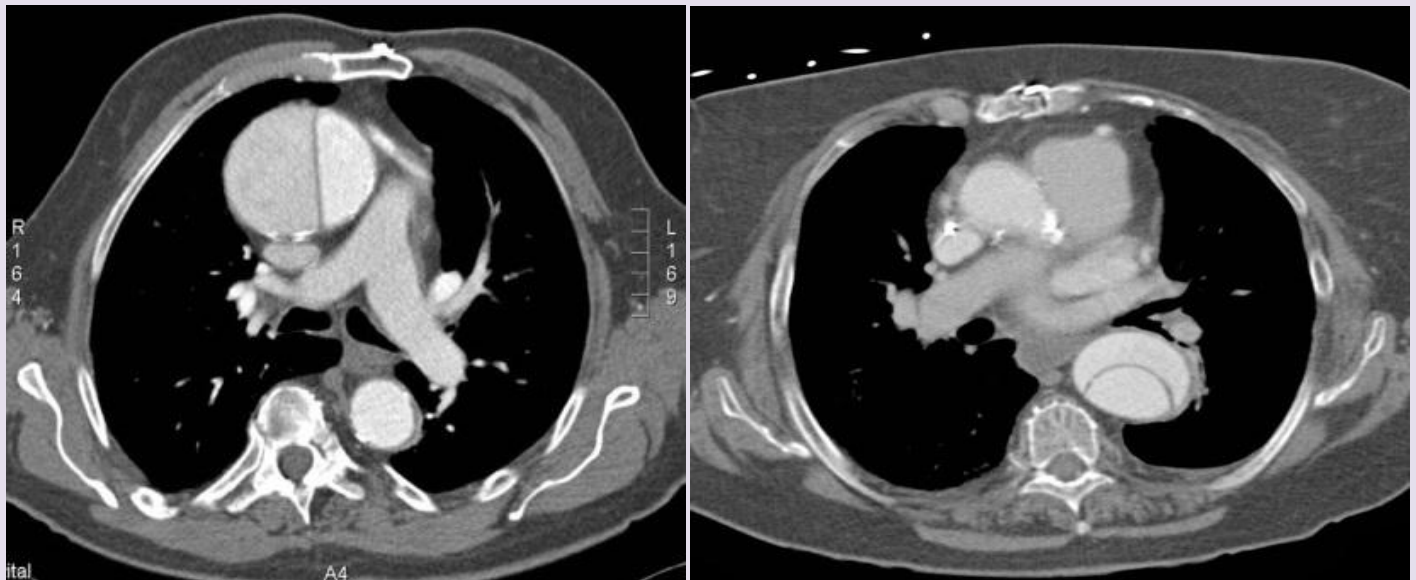


Image 1: An intraluminal tear has formed a 'flap' that can be clearly seen in the ascending aorta. This is a Stanford type A dissection

Image 2: Stanford type B dissection, seen in the descending aorta



Image 3: CT scan demonstrates an obvious flap in the thoracic aorta indicating aortic dissection. The flap is in the middle of the descending aorta (the dark line) which separates the true lumen anteriorly from the intimal flap posteriorly.

Aortic dissection vs oesophageal rupture.

Severe chest pain radiating through to the back, which is unusual in ischaemic heart disease

Features that favour oesophageal rupture over aortic dissection include:

- The history of onset **while eating**
- Blood pressure **equal in both arms**
- No diastolic murmur
- Good peripheral pulses, and
- Presence of a pleural effusion.

Pulmonary arterial hypertension (PAH)

A sustained elevation in mean pulmonary arterial pressure of **greater than 25 mmHg** at rest or **30 mmHg** after exercise

Features:

- 1) **exertional dyspnoea** is the most frequent symptom
- 2) **chest pain** and **syncope** may also occur
- 3) **loud P2**
- 4) left parasternal heave (due to right ventricular hypertrophy)

Causes and classification: PAH has recently been reclassified by the WHO:

Group 1: Pulmonary arterial hypertension (PAH)

- idiopathic*
- familial
- associated conditions:
 - 1) **Collagen vascular disease**,
 - 2) **congenital heart disease** with systemic to pulmonary shunts,
 - 3) **HIV****
 - 4) **drugs** and **toxins**
 - 5) **sickle cell disease**
- persistent pulmonary hypertension of the newborn

*previously termed primary pulmonary hypertension

**the mechanism by which HIV infection produces pulmonary hypertension remains unknown

Group 2: Pulmonary hypertension with left heart disease

- left-sided **atrial**, **ventricular** or **valvular** disease
Such as **left ventricular systolic and diastolic dysfunction**, **mitral stenosis** and **mitral regurgitation**

Group 3: Pulmonary hypertension secondary to lung disease/hypoxia

- 1) COPD
- 2) interstitial lung disease
- 3) sleep apnoea
- 4) high altitude

Group 4: Pulmonary hypertension due to thromboembolic disease

Group 5: Miscellaneous conditions

- lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis

Management of Pulmonary arterial hypertension:

- 1) Management should first involve treating any **underlying conditions**, for example with **anticoagulants** or **oxygen**.
- 2) **Acute vasodilator testing:**
 - ☒ Aims to decide which patients show a **significant fall in pulmonary arterial pressure** following the administration of vasodilators such as **intravenous epoprostenol** or **inhaled nitric oxide**
 - 1) If there is a **positive response** to acute vasodilator testing:
 - ⇒ treat with **oral calcium channel blockers**
 - 2) If there is a **negative response** to acute vasodilator testing:
 - ⇒ **prostacyclin analogues**: **treprostinil**, **iloprost**
 - ⇒ **endothelin receptor antagonists**: **bosentan**
 - ⇒ **phosphodiesterase inhibitors**: **sildenafil**

Primary pulmonary hypertension

- Primary pulmonary hypertension (**PPH**, now **IPAH**)
- pulmonary arterial pressure > **25 mmHg** at **rest**,
> **30mmHg** with **exercise**
- PPH is diagnosed when **no underlying cause** can be found
- around **10%** of cases are familial: **autosomal dominant**
- **endothelin** thought to play a key role in pathogenesis
- associated with **HIV**, **cocaine** and **anorexigens** (e.g. fenfluramine)

Features:

- 1) more common in females, typically presents at 20-40 years old
- 2) progressive SOB
- 3) cyanosis
- 4) right ventricular heave, loud P2, raised JVP with prominent 'a' waves, tricuspid regurgitation

Investigation:

- echocardiography

Management:

- 1) **diuretics** if right heart failure
- 2) **anticoagulation**
- 3) vasodilator therapy:
 - ⇒ **Calcium channel blocker**,
 - ⇒ **IV prostaglandins**,
 - ⇒ **bosentan**: endothelin-1 receptor antagonist
- 4) **heart-lung transplant**

Pericarditis

Pericarditis is one of the differentials of any patient presenting with **chest pain**.

Features:

- 1) Chest pain: may be pleuritic. Is often relieved by sitting forwards
- 2) non-productive cough, dyspnoea and flu-like symptoms
- 3) pericardial rub
- 4) tachypnoea
- 5) tachycardia

Causes:

- 1) viral infections (Coxsackie)
- 2) tuberculosis
- 3) uraemia (causes 'fibrinous' pericarditis)
- 4) trauma
- 5) post-myocardial infarction, Dressler's syndrome
- 6) connective tissue disease
- 7) hypothyroidism

ECG changes:

- 1) widespread 'saddle-shaped' ST elevation
- 2) **PR depression**: most specific ECG marker for pericarditis



ECG showing pericarditis. Note the widespread nature of the ST elevation and the PR depression

Rheumatic fever

Criteria:

- Rheumatic fever develops following an immunological reaction to recent (2-6 weeks ago) *Streptococcus pyogenes* infection.
- Diagnosis is based on evidence of recent streptococcal infection accompanied by:
 - 2 major criteria
 - 1 major with 2 minor criteria

Evidence of recent streptococcal infection:

- 1) history of scarlet fever
- 2) positive throat swab
- 3) ASOT > 200iu/mL
- 4) increase in DNase B titre

Major criteria:

- 1) erythema marginatum
- 2) subcutaneous nodules
- 3) Sydenham's chorea
- 4) polyarthritis
- 5) carditis (endo-, myo- or peri-)

Minor criteria:

- 1) raised ESR or CRP
- 2) pyrexia
- 3) arthralgia (not if arthritis a major criteria)
- 4) prolonged PR interval



Erythema marginatum is seen in around 10% of children with rheumatic fever. It is rare in adults

Infective endocarditis

- The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis.
- **The following types of patients are affected:**
 - ✓ previously normal valves (50%, typically acute presentation)
 - ✓ Rheumatic valve disease (30%)
 - ✓ prosthetic valves
 - ✓ congenital heart defects
 - ✓ intravenous drug users (IVDUs, e.g. Typically causing tricuspid lesion)

Causes:

- 1) Streptococcus **viridans** (most common cause - 40-50%)
- 2) Staphylococcus **epidermidis** (especially prosthetic valves)
- 3) Staphylococcus **aureus** (especially acute presentation, IVDUs)
- 4) Streptococcus **bovis** is associated with colorectal cancer
- 5) Streptococcus **mitis**: following dental work
- 6) **non-infective:**
 - ⇒ SLE (Libman-Sacks),
 - ⇒ malignancy: marantic endocarditis

Culture negative causes:

- 1) prior antibiotic therapy
- 2) Coxiella burnetii (Q fever)
- 3) Bartonella
- 4) Brucella
- 5) HACEK: Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella)

+ Following prosthetic valve surgery Staphylococcus epidermidis is the most common organism in the first 2 months and is usually the result of perioperative contamination.

+ After 2 months the spectrum of organisms which cause endocarditis return to normal, except with a slight increase in Staph. aureus infections

How should the blood samples be drawn to maximise the chances of obtaining positive cultures?

Draw three samples of blood from different venepuncture sites with the first separated from the last by at least one hour over 24 hours

Modified Duke Criteria:

Infective endocarditis diagnosed if:

- pathological criteria positive, or
- 2 major criteria, or
- 1 major and 3 minor criteria, or
- 5 minor criteria

Pathological criteria:

Positive histology or **microbiology** of pathological material obtained at autopsy or cardiac surgery (valve tissue, vegetations, embolic fragments or intracardiac abscess content)

Major criteria:

1) Positive blood cultures

- **two positive blood cultures** showing typical organisms consistent with infective endocarditis, such as *Streptococcus viridans* and the HACEK group, or
- **persistent bacteraemia** from two blood cultures taken > 12 hours apart or **three or more positive blood cultures** where the pathogen is less specific such as *Staph aureus* and *Staph epidermidis*, or
- **positive serology** for *Coxiella burnetii*, *Bartonella* species or *Chlamydia psittaci*, or
- **positive molecular assays** for specific gene targets

2) Evidence of endocardial involvement

- **positive echocardiogram** (oscillating structures, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves), or
- **new valvular regurgitation**

Minor criteria:

- 1) **predisposing heart condition** or **intravenous drug use**
- 2) **microbiological evidence** does not meet major criteria
- 3) **fever > 38 C**
- 4) **vascular phenomena:**
 - 1) **Major emboli,**
 - 2) **splenomegaly,**
 - 3) **clubbing,**
 - 4) **splinter haemorrhages,**
 - 5) **Janeway lesions,**
 - 6) **petechiae or purpura**
- 5) **immunological phenomena:**
 - 1) **glomerulonephritis,**
 - 2) **Osler's nodes,**
 - 3) **Roth spots**

Poor prognostic factors:

- 1) Staph aureus infection
- 2) prosthetic valve (especially 'early', acquired during surgery)
- 3) culture negative endocarditis
- 4) low complement levels

Mortality according to organism:

- staphylococci -----> **30%**
- bowel organisms ---> **15%**
- streptococci -----> **5%**

Current antibiotic guidelines (source: British National Formulary)

Scenario	Suggested antibiotic therapy
Initial blind therapy	<u>Native valve</u> • amoxicillin, consider adding low-dose gentamicin If penicillin allergic , MRSA or severe sepsis : • vancomycin + low-dose gentamicin <u>If prosthetic valve:</u> • vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by staphylococci	<u>Native valve</u> ⇒ Flucloxacillin If penicillin allergic or MRSA ⇒ vancomycin + rifampicin <u>Prosthetic valve</u> ⇒ Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA ⇒ Vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	⇒ Benzylopenicillin If penicillin allergic : ⇒ vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	⇒ Benzylopenicillin + low-dose gentamicin If penicillin allergic : ⇒ vancomycin + low-dose gentamicin

(Prosthetic valve----->.....+ rifampicin + low-dose gentamicin لازم)

Indications for surgery:

- 1) Severe valvular incompetence
- 2) Aortic abscess (often indicated by a lengthening PR interval)
- 3) infections resistant to antibiotics/fungal infections
- 4) Cardiac failure refractory to standard medical treatment
- 5) recurrent emboli after antibiotic therapy

Infective endocarditis prophylaxis:

The 2008 guidelines from NICE have radically changed the list of procedures for which antibiotic prophylaxis is recommended

NICE recommends the following procedures do not require prophylaxis:

- 1) dental procedures
- 2) upper and lower GIT procedures
- 3) genitourinary tract; this includes:
 - ⇒ urological,
 - ⇒ gynaecological and
 - ⇒ obstetric procedures and childbirth
- 4) upper and lower respiratory tract; this includes ear, nose and throat procedures and bronchoscopy

The guidelines do however suggest:

- any episodes of infection in people at risk of infective endocarditis should be investigated and treated promptly to reduce the risk of endocarditis developing
- if a person at risk of infective endocarditis is receiving antimicrobial therapy because they are undergoing a gastrointestinal or genitourinary procedure at a site where there is a suspected infection they should be given an antibiotic that covers organisms that cause infective endocarditis

Valvular Heart Disease

Heart sounds

S1:

S1 is caused by closure of **mitral** and **tricuspid** valves

Causes of a loud S1:

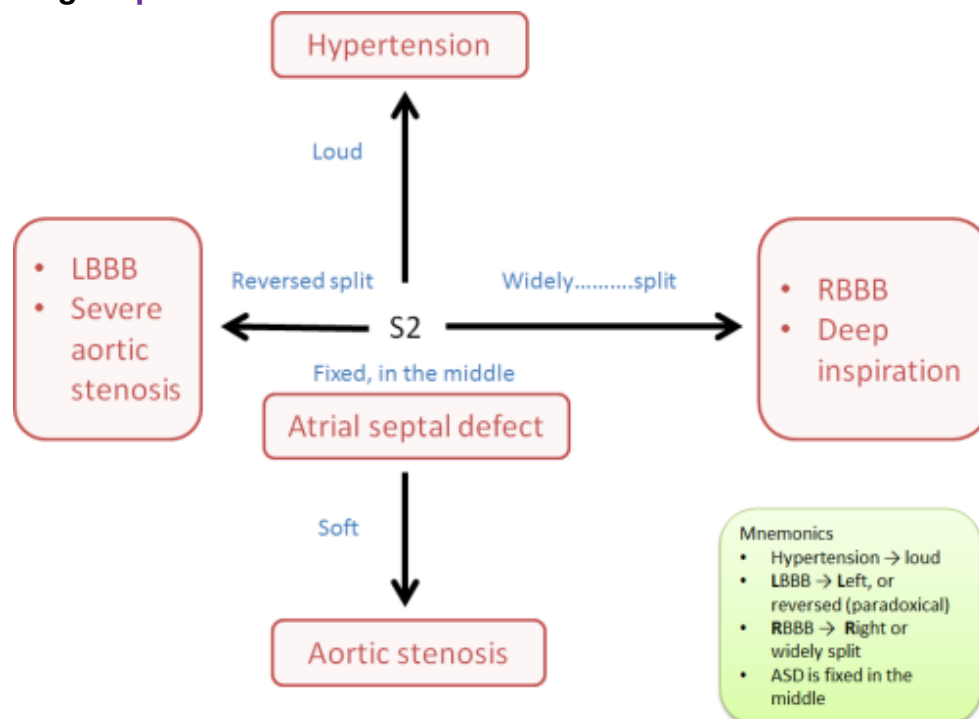
- 1) mitral stenosis
- 2) left to right shunts
- 3) short PR interval, atrial premature beats
- 4) hyperdynamic states

Causes of a quiet or soft S1:

- 1) Long PR
- 2) mitral regurgitation

S2:

- S2 is caused by the closure of the **aortic valve** (A2) closely followed by that of the **pulmonary valve** (P2)
- normally the aortic valve close before the pulmonary valve
- **soft** in aortic **stenosis**
- **splitting** during **inspiration** is normal



Causes of a loud S2:

- hypertension: **systemic** (loud A2) or **pulmonary** (loud P2)
- hyperdynamic states
- **atrial septal defect** without pulmonary hypertension

Causes of a soft S2:----->**aortic stenosis**

Causes of fixed split S2:

- atrial septal defect
- vsd

Causes of a widely split S2: (the pulmonary valve takes long time to close)

- deep inspiration
- RBBB
- pulmonary stenosis
- severe mitral regurgitation

Causes of a reversed (paradoxical) split S2 (P2 occurs before A2):

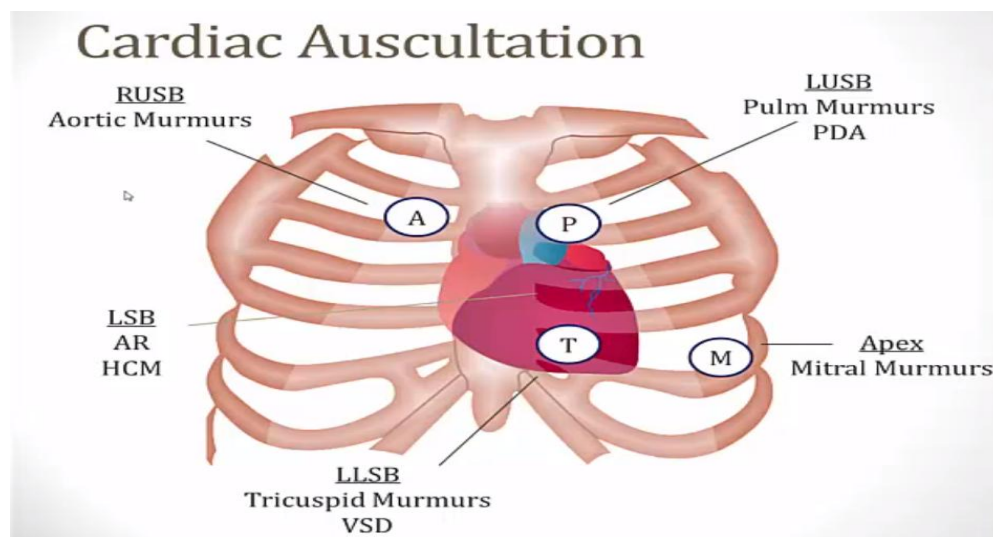
- LBBB
- severe aortic stenosis
- right ventricular pacing
- WPW type B (causes early P2)
- patent ductus arteriosus

S3 (third heart sound)

- Caused by **diastolic filling of the ventricle**
- considered normal if < 30 years old (may persist in women up to 50 years old {oestrogen may cause relaxation of muscles})
- heard in:
 - ⇒ **Left ventricular failure** (e.g. dilated cardiomyopathy),
 - ⇒ **constrictive pericarditis** (called a pericardial knock)

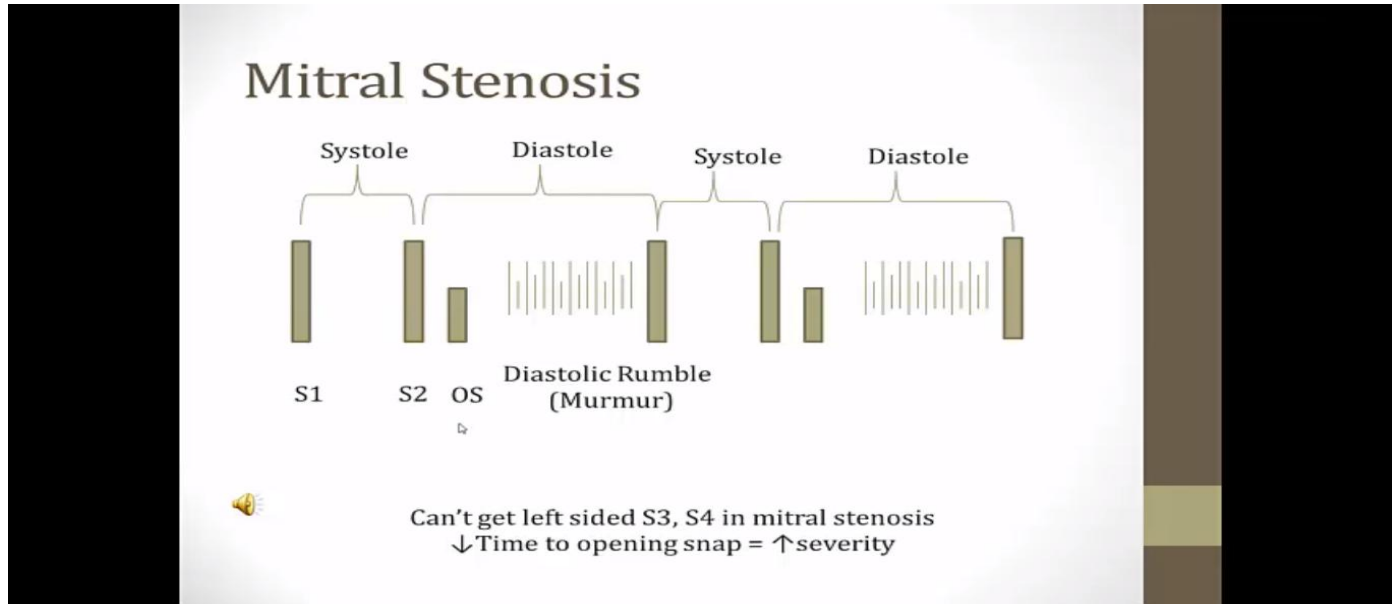
S4 (fourth heart sound):

- Caused by **atrial contraction against a stiff ventricle**
- may be heard in:
 - ⇒ **aortic stenosis,**
 - ⇒ **HOCM,**
 - ⇒ **hypertension**
- in **HOCM** a double apical impulse may be felt as a result of a palpable S4



Mitral stenosis

- The causes of mitral stenosis are rheumatic fever, rheumatic fever and rheumatic fever.
- Rarer causes that may be seen in the exam include:
 - 1) mucopolysaccharidoses,
 - 2) carcinoid and
 - 3) endocardial fibroelastosis



Features:

- mid-late diastolic murmur (best heard in expiration)
- loud S1, opening snap
- low volume pulse
- malar flush
- atrial fibrillation

Features of severe MS:

- length of murmur increases
- opening snap becomes closer to S2

Echocardiography:

- The normal cross sectional area of the mitral valve is 4-6 sq cm.
- A 'tight' mitral stenosis implies a cross sectional area of < 1 sq cm

The severity of mitral stenosis can be graded:

Severity of mitral stenosis	Severity Valve area (cm ²)	Gradient (mmHg)
Mild	1.6-2.0	<5
Moderate	1.0-1.5	5-10
Severe	<1.0	>10

Mitral valve prolapse

- Mitral valve prolapse is common, occurring in around 5-10 % of the population.
- It is usually idiopathic but may be associated with a wide variety of cardiovascular disease and other conditions

Associations:

- 1) congenital heart disease: PDA, ASD
- 2) cardiomyopathy
- 3) long-QT syndrome
- 4) Wolff-Parkinson White syndrome
- 5) Turner's syndrome
- 6) Marfan's syndrome, Fragile X
- 7) Ehlers-Danlos Syndrome
- 8) osteogenesis imperfecta
- 9) pseudoxanthoma elasticum
- 10) polycystic kidney disease

Features:

- patients may complain of atypical chest pain or palpitations
- mid-systolic click (occurs later if patient squatting)
- late systolic murmur (longer if patient standing)

Complications:

- 1) Mitral regurgitation,
- 2) arrhythmias (including long QT),
- 3) emboli,
- 4) sudden death

Aortic stenosis

Causes of aortic stenosis:

- 1) Degenerative calcification (most common cause in older patients > 65 years)
- 2) bicuspid aortic valve (most common cause in younger patients < 65 years)
- 3) William's syndrome (supravalvular aortic stenosis)
- 4) HOCM: subvalvular
- 5) post-rheumatic disease

Features of severe aortic stenosis:

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Management:

- 1) if asymptomatic then observe the patient is general rule
- 2) if symptomatic then valve replacement
- 3) if asymptomatic but valvular gradient > 50 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- 4) balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement

A guide to determining the severity of aortic stenosis is given below:

Severity of aortic stenosis	Severity Valve area (cm ²)	Mean gradient (mmHg)
Mild	> 1.5	< 25
Moderate	1.0 - 1.5	25 - 50
Severe	< 1.0	> 50
Critical	< 0.7	> 80

Aortic regurgitation

Causes of acute aortic insufficiency:

- Rheumatic
- Infective endocarditis
- Ruptured sinus of Valsalva
- Trauma, prosthetic valve surgery, and
- Aortic dissection, laceration of the aorta.

Causes of chronic aortic insufficiency:

- Rheumatic
- Syphilis
- Aortitis (that is, Takayasu disease)
- Marfan's syndrome
- Osteogenesis imperfecta
- Bicuspid aortic valve, defect of the interventricular septum or sinus of Valsalva
- Ankylosing spondylitis
- Reiter's syndrome
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Hypertension, and
- Infective endocarditis.

Causes due to valve disease	Causes due to aortic root disease
1) rheumatic fever 2) infective endocarditis 3) connective tissue diseases e.g. RA/SLE 4) bicuspid aortic valve	1) aortic dissection 2) spondylarthropathies (e.g. ankylosing spondylitis) 3) hypertension 4) syphilis 5) Marfan's, Ehler-Danlos syndrome

Features

- early diastolic murmur
- collapsing pulse
- wide pulse pressure
- mid-diastolic Austin-Flint murmur in severe AR
⇒ due to partial closure of the anterior mitral valve cusps caused by the regurgitation streams

Associated clinical signs that accompany aortic regurgitation include:

- 1) **Wide pulse pressure**
- 2) **Decrescendo early diastolic murmur** (heard best while the patient is leaning forward on deep expiration)
- 3) **Austin-Flint murmur** (caused by the regurgitant flow causing vibration of the mitral apparatus)
- 4) **Pulsus bisferiens**; increased pulse pressure; visible, forceful, and bounding peripheral pulses (water hammer)
- 5) **Corrigan's pulse** - Quickly collapsing pulse
- 6) **Musset's sign** - Bobbing of the head
- 7) **Quincke's sign** - Capillary pulsations of the nail bed
- 8) **Muller's sign** - Pulsations of the uvula
- 9) **Traube's sign** - Loud systolic sound over femoral arteries ('pistol-shot' femorals)
- 10) **Duroziez sign** - Systolic-diastolic murmur produced by compression of femoral artery with a stethoscope.

Bicuspid aortic valve

Overview:

- occurs in 1-2% of the population
- usually asymptomatic in childhood
- the majority eventually develop aortic stenosis or regurgitation
- associated with:
 - 1) **A left dominant coronary circulation** (the posterior descending artery arises from the circumflex instead of the right coronary artery) and
 - 2) **Turner's syndrome**
- around 5% of patients also have **coarctation of the aorta**

Complications:

- aortic stenosis/regurgitation as above
- higher risk for aortic dissection and aneurysm formation of the ascending aorta

Tricuspid regurgitation

Causes:

- 1) right ventricular dilation
- 2) pulmonary hypertension e.g. COPD
- 3) rheumatic heart disease
- 4) infective endocarditis (especially intravenous drug users)
- 5) Ebstein's anomaly
- 6) carcinoid syndrome

Signs:

- pan-systolic murmur
- giant V waves in JVP
- pulsatile hepatomegaly
- left parasternal heave

Atrial myxoma

- 75% occur in **left atrium**
- more common in **females**

Features:

- 1) systemic: dyspnoea, fatigue, weight loss, fever, clubbing
- 2) emboli
- 3) atrial fibrillation
- 4) mid-diastolic murmur, 'tumour plop'
- 5) echo: pedunculated heterogeneous mass typically attached to the fossa ovalis region of the interatrial septum

Murmurs

Ejection systolic:

- aortic stenosis, HOCM
- pulmonary stenosis,
- ASD, Fallot's

Holosystolic (pansystolic)

- mitral/tricuspid regurgitation (high-pitched and 'blowing' in character)
- VSD ('harsh' in character)

Late systolic:

- mitral valve prolapse
- coarctation of aorta

Early diastolic:

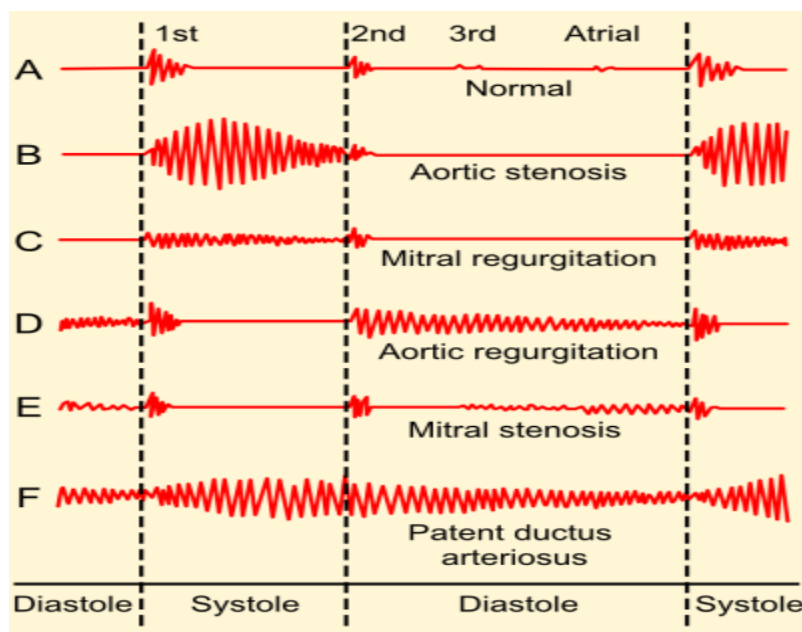
- aortic regurgitation (high-pitched and 'blowing' in character)
- Graham-Steel murmur (pulmonary regurgitation, again high-pitched and 'blowing' in character)

Mid-late diastolic:

- mitral stenosis ('rumbling' in character)
- Austin-Flint murmur (severe aortic regurgitation, again is 'rumbling' in character)

Continuous machine-like mumur:

- patent ductus arteriosus



Pulses

Pulsus paradoxus:

- greater than the normal (10 mmHg) fall in systolic blood pressure during inspiration → faint or absent pulse in inspiration
- severe asthma, cardiac tamponade

Slow-rising/plateau:

- aortic stenosis

Collapsing:

- aortic regurgitation
- patent ductus arteriosus
- hyperkinetic (anaemia, thyrotoxic, fever, exercise/pregnancy)

Pulsus alternans:

- regular alternation of the force of the arterial pulse
- severe LVF

Bisferiens pulse: 'double pulse' - two systolic peaks

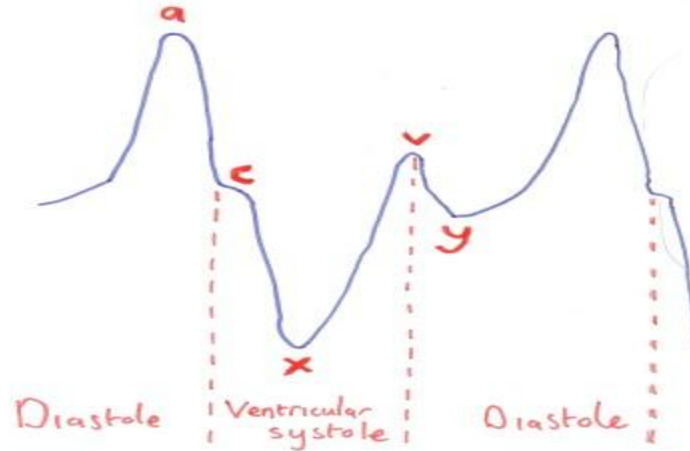
- mixed aortic valve disease
- HOCM may occasionally be associated with a bisferiens pulse

Jerky pulse:

- hypertrophic obstructive cardiomyopathy

Jugular venous pulse

- As well as providing information on right atrial pressure, the jugular vein waveform may provide clues to underlying valvular disease.
- **Non-pulsatile JVP** is seen in **superior vena caval obstruction**.
- **Kussmaul's sign** describes a **paradoxical rise in JVP during inspiration** seen in **constrictive pericarditis**.
- **Constrictive pericarditis** produces an **elevated JVP**, with **prominent x** and **y descent**



'a' wave = atrial contraction

- large if atrial pressure e.g. tricuspid stenosis, pulmonary stenosis, pulmonary hypertension
- absent if in atrial fibrillation

Cannon 'a' waves:

- caused by atrial contractions against a closed tricuspid valve
- are seen in:
 - ✓ Complete heart block,
 - ✓ ventricular tachycardia/ectopics,
 - ✓ nodal rhythm,
 - ✓ single chamber ventricular pacing

'c' wave:

- closure of tricuspid valve
- not normally visible

'v' wave:

- due to passive filling of blood into the atrium against a closed tricuspid valve
- giant v waves in **tricuspid regurgitation**

'x' descent = fall in atrial pressure during ventricular systole

'y' descent = opening of tricuspid valve

Constrictive pericarditis produces an elevated JVP, with prominent x and y descent

Cannon waves

- Caused by the right atrium contracting against a closed tricuspid valve.
- May be subdivided into regular or intermittent

Regular cannon waves:

- ventricular tachycardia (with 1:1 ventricular-atrial conduction)
- atrio-ventricular nodal re-entry tachycardia (AVNRT)

Irregular cannon waves:

- complete heart block

Prosthetic heart valves

- The most common valves which need replacing are the aortic and mitral valve.
- There are two main options for replacement: biological (bioprosthetic) or mechanical.

Biological (bioprosthetic) valves	Mechanical valves
• Usually bovine or porcine in origin • Major disadvantage is structural deterioration and calcification over time. • Most older patients (> 65 years for aortic valves and > 70 years for mitral valves) receive a bioprosthetic valve • Long-term anticoagulation not usually needed. • Warfarin may be given for the first 3 months depending on patient factors. • Low-dose aspirin is given long-term.	• The most common type now implanted is the bileaflet valve. • Ball-and-cage valves are rarely used nowadays • Mechanical valves have a low failure rate • Major disadvantage is the increased risk of thrombosis meaning long-term anticoagulation is needed. • Aspirin is normally given in addition unless there is a contraindication. <u>Target INR:</u> • aortic: 2.0-3.0 • mitral: 2.5-3.5

Congenital Heart Disease

Types:

Acyanotic - most common causes

- 1) ventricular septal defects (VSD) - most common, accounts for 30%
- 2) atrial septal defect (ASD)
- 3) patent ductus arteriosus (PDA)
- 4) coarctation of the aorta
- 5) aortic valve stenosis

VSDs are more common than ASDs. However, in adult patients ASDs are the more common new diagnosis as they generally presents later

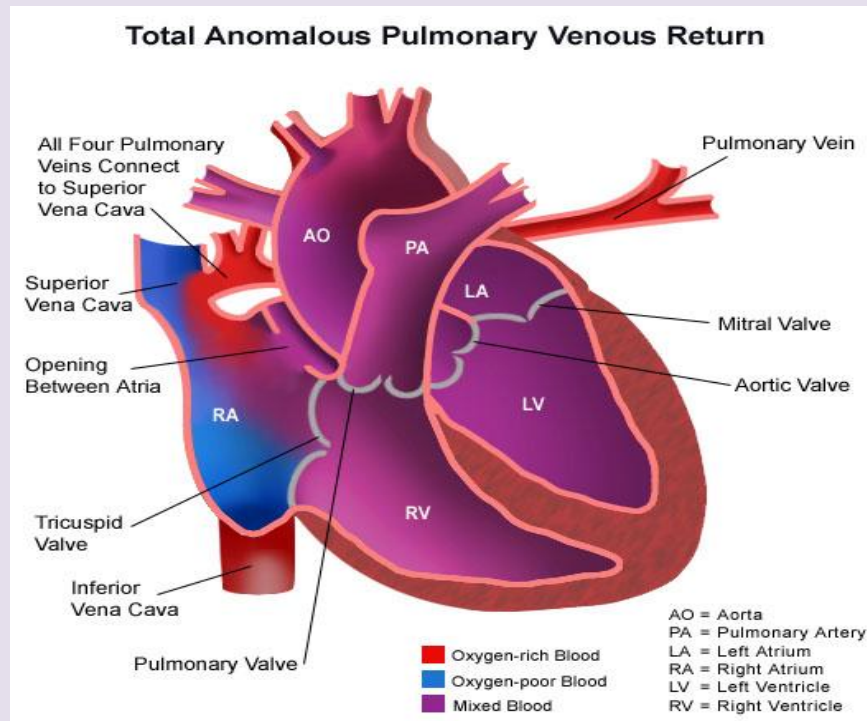
Cyanotic - most common causes

- 1) tetralogy of Fallot
- 2) transposition of the great arteries (TGA)
- 3) tricuspid atresia
- 4) pulmonary valve stenosis

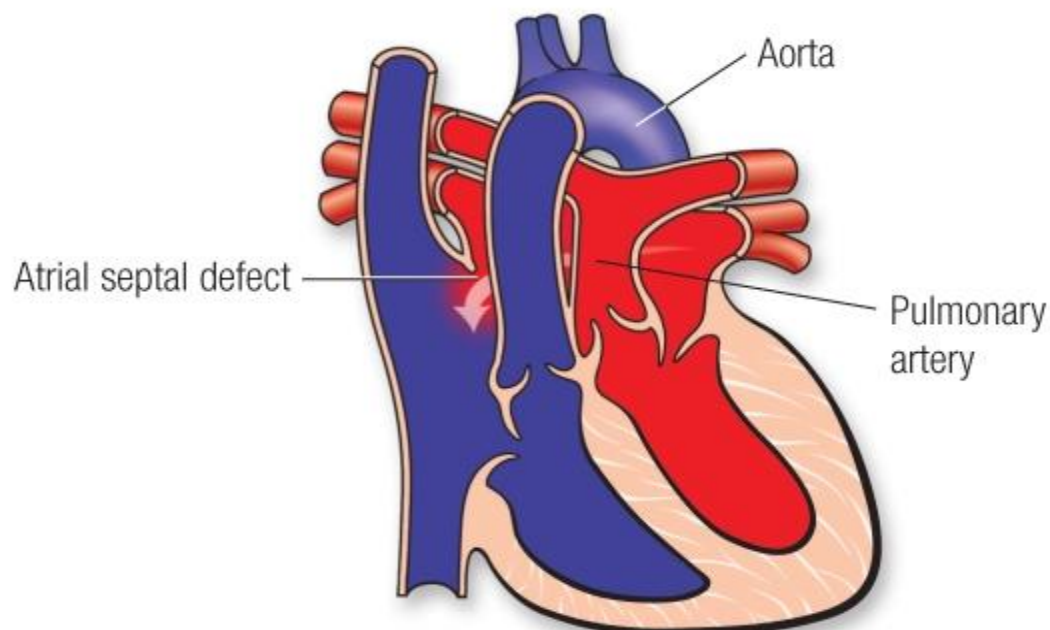
Fallot's is more common than TGA. However, at birth TGA is the more common lesion as patients with Fallot's generally presenting at around 1-2 months

Total anomalous pulmonary venous drainage (TAPVD)

- associated with **cyanosis** in the newborn
- Total anomalous pulmonary venous connection (TAPVC) consists of an abnormality of blood flow in which **all 4 pulmonary veins drain into systemic veins or the right atrium** with or without pulmonary venous obstruction.
- Systemic and pulmonary venous blood mix in the right atrium



Transposition of the Great Arteries



Ventricular septal defects

- VSDs are the most common cause of congenital heart disease.
- They close spontaneously in around 50% of cases.
- **Congenital VSDs** are associated with:
 - 1) Chromosomal disorders (e.g. Down's syndrome, Edward's syndrome, Patau syndrome) and
 - 2) Single gene disorders such as
- **Non-congenital** causes include post myocardial infarction

Features:

- classically a pan-systolic murmur which is louder in smaller defects

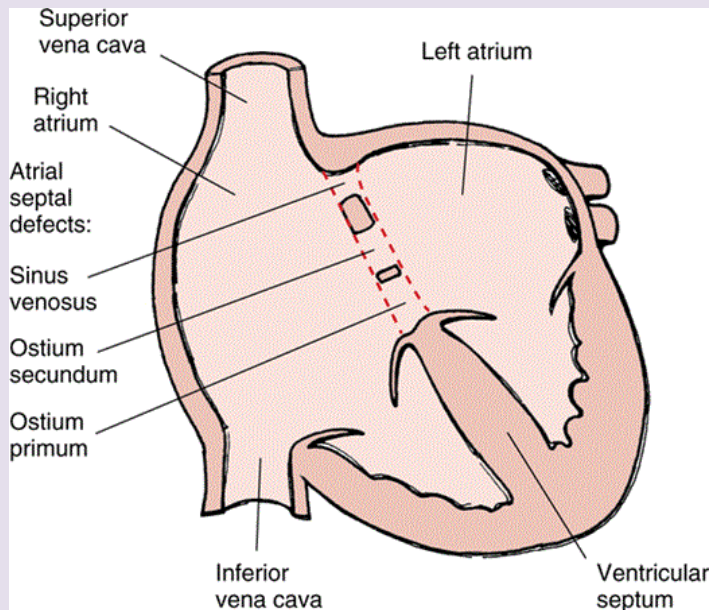
Complications:

- aortic regurgitation*
- infective endocarditis
- Eisenmenger's complex
- right heart failure
- pulmonary hypertension: pregnancy is contraindicated in women with pulmonary hypertension as it carries a 30-50% risk of mortality

*aortic regurgitation is due to a poorly supported right coronary cusp resulting in cusp prolapse

Atrial septal defects

- Atrial septal defects (ASDs) are the most likely congenital heart defect to be found in adulthood.
- They carry a significant mortality, with 50% of patients being dead at 50 years.



- Two types of ASDs are recognised,
 - ✓ **ostium primum**
 - ✓ **ostium secundum** (the most common)

Features:

- ejection systolic murmur,
- fixed splitting of S2
- embolism may pass from venous system to left side of heart causing a stroke

Ostium primum:

- present earlier than ostium secundum defects
- associated with abnormal AV valves
- ECG: **RBBB** with **LAD**, **prolonged PR interval**

Ostium secundum (70% of ASDs)

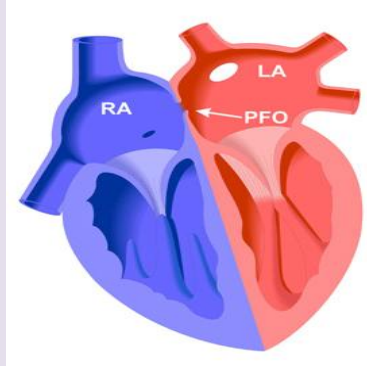
- associated with Holt-Oram syndrome (tri-phalangeal thumbs)
- ECG: **RBBB** with **RAD**

Ostium primum: ECG: RBBB with LAD, prolonged PR interval

Left (L----P)..... (R----S)

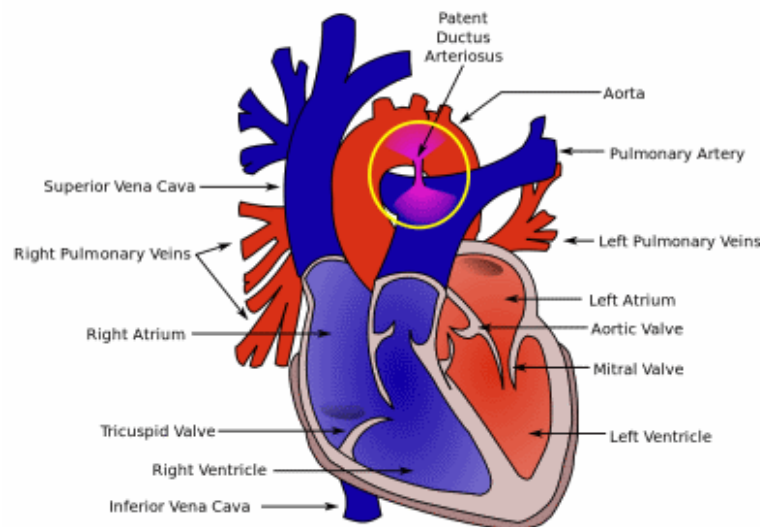
Patent foramen ovale

- Patent foramen ovale (PFO) is present in around 20% of the population.
- It may allow embolus (e.g. from DVT) to pass from right side of the heart to the left side leading to a stroke - 'a paradoxical embolus'
- There also appears to be an association between **migraine** and PFO.
- Some studies have reported improvement in migraine symptoms following closure of the PFO



Patent ductus arteriosus

- acyanotic congenital heart defect
- connection between the pulmonary trunk and descending aorta
- more common in premature babies, born at high altitude or maternal rubella infection in the first trimester



Features:

- left subclavicular thrill
- continuous '**machinery**' murmur
- large volume, bounding, collapsing pulse
- wide pulse pressure
- heaving apex beat

Management:

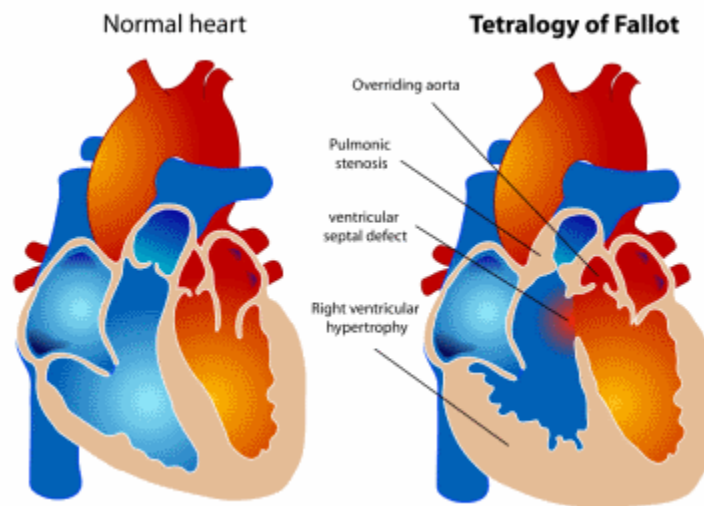
- 1) **indomethacin** closes the connection in the majority of cases
- 2) if associated with another congenital heart defect amenable to surgery then **prostaglandin E1** is useful to keep the duct open until after surgical repair

Tetralogy of Fallot

- TOF is the most common cause of cyanotic congenital heart disease.
- However, at birth transposition of the great arteries is the more common lesion as patients with TOF generally present at around 1-2 months
- It typically presents at around 1-2 months, although may not be picked up until the baby is 6 months old
- TOF is a result of anterior malalignment of the aorticopulmonary septum.

The four characteristic features are:

- 1) **overriding aorta**
- 2) **pulmonary stenosis (PS)**: right ventricular outflow tract obstruction,
- 3) **ventricular septal defect (VSD)**
- 4) **right ventricular hypertrophy(RVH)**



- The severity of the right ventricular outflow tract obstruction determines the degree of cyanosis and clinical severity

Other features:

- 1) cyanosis
- 2) causes a right-to-left shunt
- 3) ejection systolic murmur due to pulmonary stenosis (the VSD doesn't usually cause a murmur)
- 4) a right-sided aortic arch is seen in 25% of patients
- 5) chest x-ray shows a 'boot-shaped' heart,
- 6) ECG shows right ventricular hypertrophy

Management:

- 1) **surgical repair** is often undertaken in two parts
- 2) cyanotic episodes may be helped by **beta-blockers** to reduce infundibular spasm

The most common residual lesion in repaired tetralogy of Fallot is **pulmonary regurgitation**.

Eisenmenger's syndrome

- The **reversal of a left-to-right shunt** in a congenital heart defect due to **pulmonary HTN**.
- This occurs when an uncorrected left-to-right leads to **remodeling of the pulmonary microvasculature**, eventually causing obstruction to pulmonary blood and **pulmonary hypertension**.

Associated with:

- 1) ventricular septal defect
- 2) atrial septal defect
- 3) patent ductus arteriosus

Features:

- 1) original murmur may disappear
- 2) cyanosis
- 3) clubbing
- 4) **right ventricular failure**
- 5) haemoptysis, embolism

Management:

- heart-lung transplantation is required

Ebstein's anomaly

- A congenital heart defect characterised by **low insertion of the tricuspid valve** resulting in a large atrium and small ventricle.
- It is sometimes referred to as '**atrialisation**' of the **right ventricle**.
- Ebstein's anomaly may be caused by exposure to **lithium** in-utero

Associations:

- 1) **tricuspid incompetence** (pan-systolic murmur, giant V waves in JVP)
- 2) **Wolff-Parkinson White syndrome**

Cardiac imaging

Non-invasive techniques excluding echocardiography

The ability to image the heart using non-invasive techniques such as MRI, CT and radionuclides has evolved rapidly over recent years.

Nuclear imaging:

- These techniques use radiotracers which are extracted by normal myocardium.
- Examples include:
 - 1) **thallium**
 - 2) **technetium (99mTc) sestamibi**: a coordination complex of the radioisotope technetium-99m with the ligand methoxyisobutyl isonitrile (MIBI), used in 'MIBI' or cardiac Single Photon Emission Computed Tomography (SPECT) scans
 - 3) **fluorodeoxyglucose (FDG)**: used in Positron Emission Tomography (PET) scans
- The primary role of **SPECT** is to assess **myocardial perfusion** and **myocardial viability**.
- Two sets of images are usually acquired:
 - ⇒ First the myocardium at rest
 - ⇒ Followed by images of the myocardium during stress (either exercise or following adenosine / dipyridamole).
- By comparing the rest with stress images any areas of ischaemia can be classified as reversible or fixed (e.g. following a myocardial infarction).
- **Cardiac PET** is predominately a research tool at the current time

MUGA: Multi Gated Acquisition Scan

- also known as **radionuclide angiography**
- radionuclide (technetium-99m) is injected intravenously
- the patient is placed under a gamma camera
- may be performed as a stress test
- Can accurately measure **left ventricular ejection fraction**.
- **Typically used before and after cardiotoxic drugs are used**

Cardiac Computed Tomography (CT)

- Cardiac CT is useful for assessing suspected **ischaemic heart disease**,
- uses two main methods:
 - 1) **Calcium score**:
 - ✓ There is known to be a correlation between the amount of atherosclerotic plaque calcium and the risk of future ischaemic events.
 - ✓ Cardiac CT can quantify the amount of calcium producing a 'calcium score'
 - 2) **Contrast enhanced CT**: allows visualisation of the **coronary artery lumen**
- If these two techniques are combined cardiac CT has **a very high negative predictive value** for ischaemic heart disease.

Cardiac MRI : (CMR)

- Cardiac MRI has become the **gold standard** for providing **structural images of the heart**.
- It is particularly useful when:
 - 1) assessing congenital heart disease,
 - 2) determining right and left ventricular mass and
 - 3) Differentiating forms of cardiomyopathy.
- **Myocardial perfusion** can also be assessed following the administration of **gadolinium**.
- Currently CMR provides limited data on the extent of coronary artery disease.

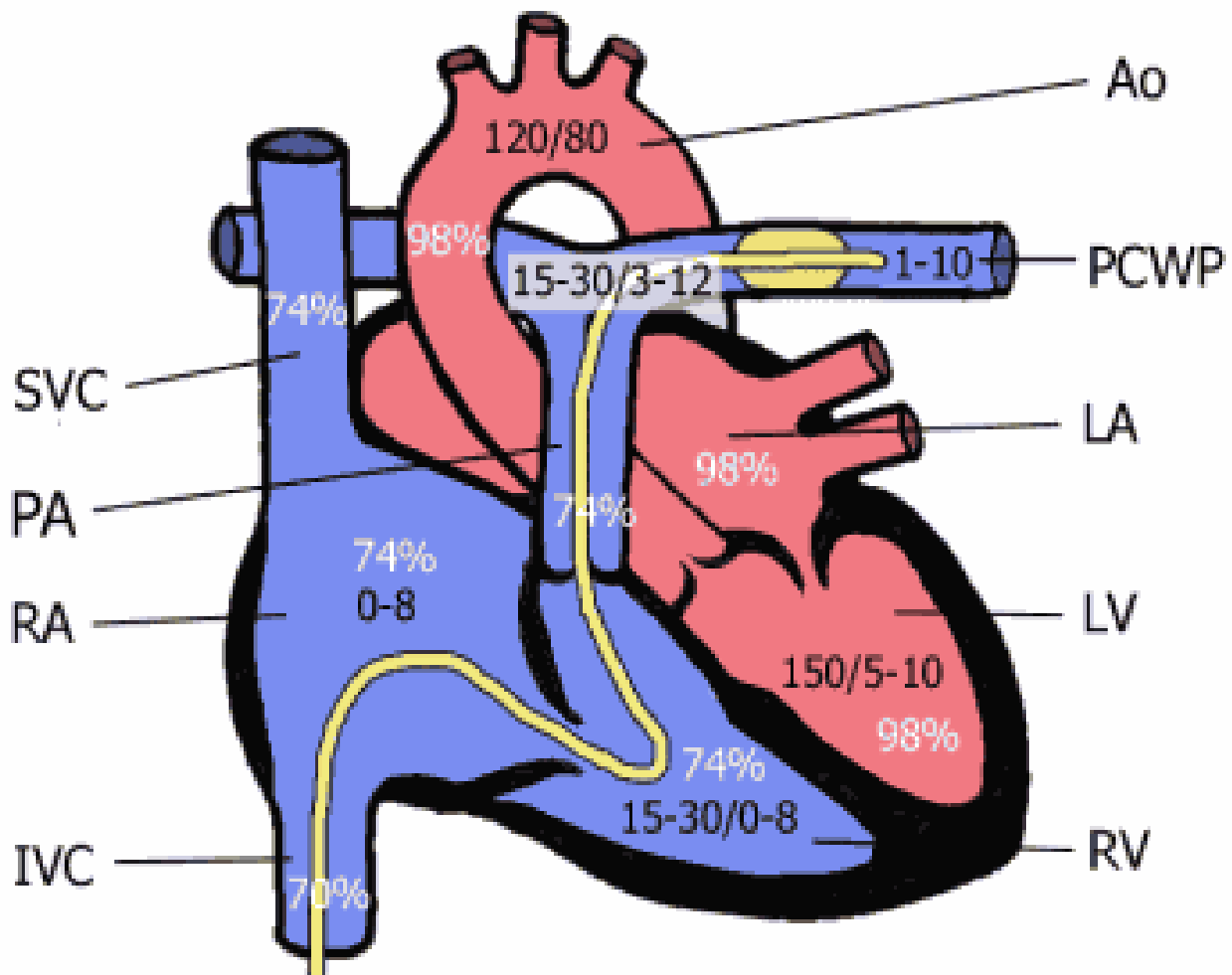
Please also see the British Heart Foundation link for an excellent summary.

Cardiac catheterisation and oxygen saturation levels

- Questions regarding cardiac catheterisation and oxygen saturation levels can seem daunting at first but a few simple rules combined with logical deduction can usually produce the answer.
- Let's start with the basics:
 - ✓ Deoxygenated blood returns to the right side of the heart via the superior vena cava (SVC) and inferior vena cava (IVC).
 - ✓ It has an oxygen saturation level of around 70%.
 - ✓ The right atrium (RA), right ventricle (RV) and pulmonary artery (PA) normally have oxygen saturation levels of around 70%
 - ✓ The lungs oxygenate the blood to a level of around 98-100%.
 - ✓ The left atrium (LA), left ventricle (LV) and aorta should all therefore have oxygen saturation levels of 98-100%

Some examples:

Diagnosis & notes	RA	RV	PA	LA	LV	Aorta
Normal	70%	70%	70%	100%	100%	100%
Atrial septal defect (ASD) The oxygenated blood in the LA mixes with the deoxygenated blood in the RA, resulting in intermediate levels of oxygenation from the RA onwards	85%	85%	85%	100%	100%	100%
Ventricular septal defect (VSD) The oxygenated blood in the LV mixes with the deoxygenated blood in the RV, resulting in intermediate levels of oxygenation from the RV onwards. The RA blood remains deoxygenated	70%	85%	85%	100%	100%	100%
Patent ductus arteriosus (PDA) Remember, a PDA connects the higher pressure aorta with the lower pressure PA. This results in only the PDA having intermediate oxygenation levels	70%	70%	85%	100%	100%	100%
VSD with Eisenmenger's	70%	70%	70%	100%	85%	85%
PDA with Eisenmenger's	70%	70%	70%	100%	100%	85%
ASD with Eisenmenger's	70%	70%	70%	85%	85%	85%



Cardiac catheter data

Normal cardiac pressures & oxygen saturation

	End systolic (mmHg)	End diastolic (mmHg)	Mean	O ₂ saturation
Left heart				
Aorta	120	80	95	98%
LV	150	5-10		98%
LA				98%
Right heart				
RA			0-8	74%
RV	15-30	0-8		74%
PA	15-30	3-12	9-16	74%
SVC				74%
IVC				70%
PCWP			1-10	

Angiodysplasia

- A vascular deformity of the gastrointestinal tract which predisposes to bleeding and iron deficiency anaemia.
- There is thought to be an association with **aortic stenosis**, although this is debated.
- Angiodysplasia is generally seen in elderly patients

Diagnosis:

- 1) colonoscopy
- 2) mesenteric angiography if acutely bleeding

Management:

- 1) endoscopic cautery or argon plasma coagulation
- 2) antifibrinolytics e.g. Tranexamic acid
- 3) oestrogens may also be used

Cardiovascular disorders

The guidelines below relate to car/motorcycle use unless specifically stated. For obvious reasons, the rules relating to drivers of heavy goods vehicles tend to be much stricter

Specific rules:

- 1) **successful catheter ablation for an arrhythmia** -----> **2 days off driving**
- 2) **angioplasty (elective)** -----> **1 week off driving**
- 3) **pacemaker insertion:** -----> **1 week off driving**
- 4) **CABG** -----> **4 weeks off driving**
- 5) **acute coronary syndrome**-----> **4 weeks off driving,**
If successfully treated by angioplasty → **1 week**
- 6) **Implantable cardioverter-defibrillator (ICD):**
 - ✓ If implanted for sustained ventricular arrhythmia: --> cease driving for **6 months**.
 - ✓ If implanted prophylactically -----> then cease driving for **1 month**.
 - ✓ Having an ICD results in -----> a permanent bar for Group 2 drivers
- 7) **Hypertension:**
 - ⇒ Can drive unless treatment causes unacceptable side effects, no need to notify DVLA.
 - ⇒ If Group 2 Entitlement the disqualifies from driving if resting BP consistently 180 mmHg systolic or more and/or 100 mm Hg diastolic or more
- 8) **angina:** -----> **driving must cease if symptoms occur at rest/at the wheel**
- 9) **Aortic aneurysm of 6cm or more** --> **notify DVLA.**
Licensing will be permitted subject to annual review.
- 10) **An aortic diameter of 6.5 cm or more:** --> **disqualifies patients from driving**
- 11) **heart transplant: DVLA do not need to be notified**

- 1) successful catheter ablation----> 2 days
- 2) angioplasty (elective or after ACS), pacemaker insertion----> 1wk off driving
- 3) CABG or ACS---> 4 weeks off driving
- 4) angina - driving must cease if symptoms occur at rest/at the wheel
- 5) ICD (for sustained Ve Tac)----> 6 month, prophylactic: 1 month
- 6) AA of 6cm or more - notify DVLA.. if 6.5 cm or more disqualifies from driving
- 7) heart transplant: DVLA do not need to be notified
- 8) HTN.....

Adult advanced life support

The joint European Resuscitation Council and Resuscitation Council (UK) 2010 guidelines do not alter significantly from the 2005 guidelines. Please see the link for more details, below is only a very brief summary of key points / changes.

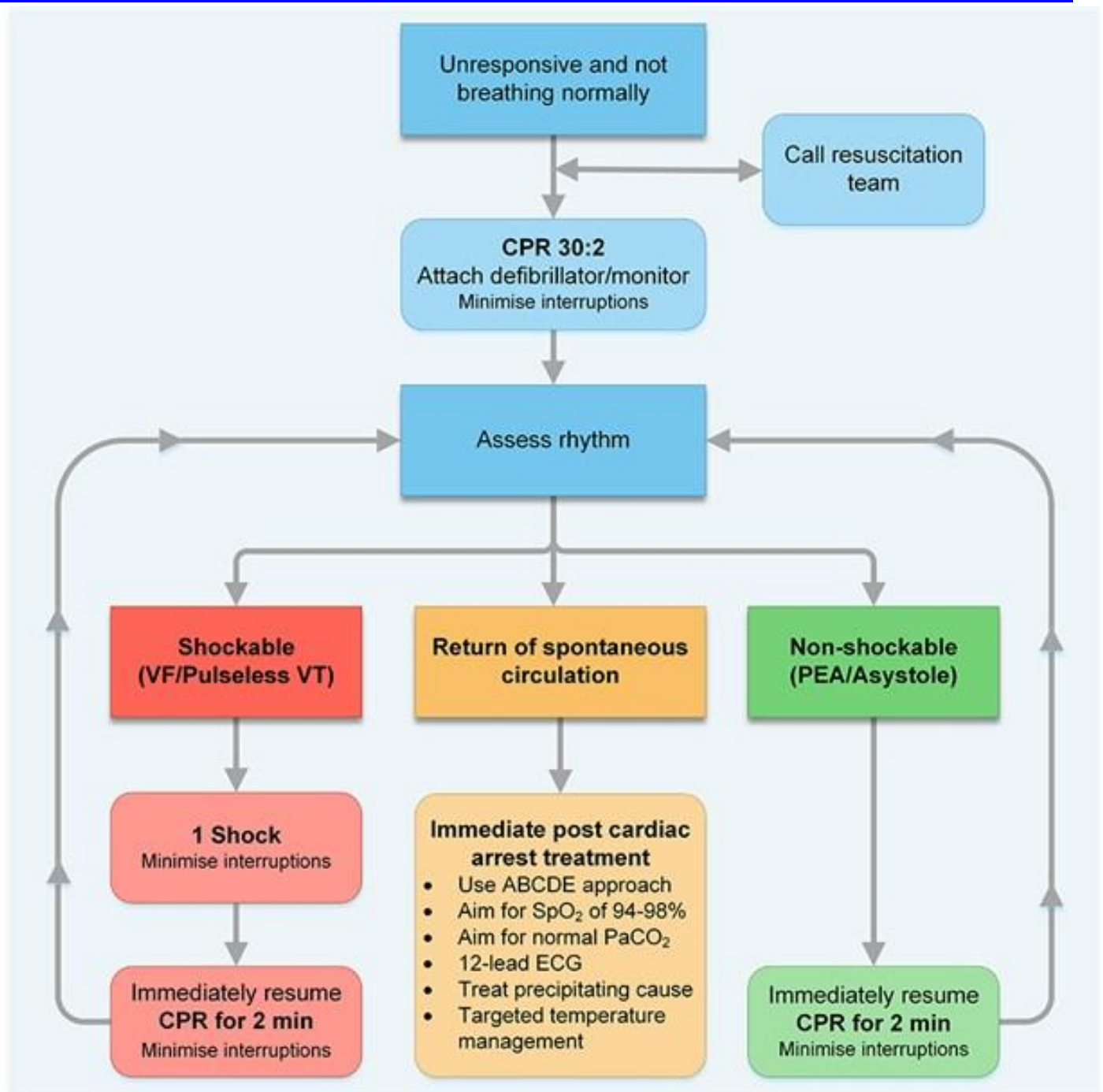
Major points include:

- 1) ratio of chest compressions to ventilation is **30:2**
- 2) chest compressions are now continued **while a defibrillator is charged**
- 3) **During a VF/VT cardiac arrest, adrenaline 1 mg** is given once chest compressions have restarted after the third shock and then every 3-5 minutes (during alternate cycles of CPR).
- 4) **Amiodarone 300 mg** is also given after the third shock (after adrenaline)
 - ⇒ Give amiodarone 300 mg IV after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest.
 - ⇒ Consider a further dose of **amiodarone 150 mg IV** after a total of five defibrillation attempts.
 - ⇒ **Lidocaine 1 mg kg** may be used as an alternative if amiodarone is not available but do not give lidocaine if amiodarone has been given already.
- 5) Atropine is **no longer recommended** for routine use in **asystole** or **pulseless electrical activity** (PEA).
- 6) **a single shock** for VF/pulseless VT followed by **2 minutes of CPR**, rather than a series of 3 shocks followed by 1 minute of CPR
- 7) asystole/pulseless-electrical activity should be treated with **2 minutes of CPR**, rather than 3, prior to reassessment of the rhythm
- 8) ~~delivery of drugs via a tracheal tube~~ is **no longer recommended**
- 9) Following successful resuscitation oxygen should be titrated to achieve saturations of 94-98%. This is to address the potential harm caused by hyperoxaemia

<https://www.resus.org.uk/resuscitation-guidelines/adult-advanced-life-support/>

The guidelines recommend the first dose of adrenaline (1 mg) after the third shock, but after chest compressions have restarted immediately.

The Adult advanced life support algorithm has been modified slightly to show 2015 changes.



During CPR

- Ensure high quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

Treat Reversible Causes

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Thrombosis - coronary or pulmonary
- Tension pneumothorax
- Tamponade – cardiac
- Toxins

Consider

- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

Post-cardiac arrest syndrome

The 2010 UK Resuscitation Guidelines describe care after a successful cardiac arrest, and how to minimise the complications of the "post-cardiac arrest syndrome".

- 1) Maintain glucose **<10 mmol/L**.
- 2) Hyperglycaemia and hypoglycaemia are both associated with adverse outcomes and should be actively avoided.
- 3) Trials investigating tight sugar control (4.5-6.0 mmol/L) demonstrated worse outcomes (due to increased hypoglycaemia).
- 4) Hyperoxaemia (and hypoxia) is also associated with poor outcomes, so oxygen saturations should be kept at **94-98%**, not 100%.
- 5) Hyperpyrexia is a poor sign but this should be treated reactively, not prophylactically. This is also the case for seizures.

Syncope

- A transient loss of consciousness due to global cerebral hypoperfusion with rapid onset, short duration and spontaneous complete recovery
- Note how this definition excludes other causes of collapse such as epilepsy.

The European Society of Cardiology published guidelines in 2009 on the investigation and management of syncope.

They suggested the following classification:

- 1) Reflex syncope (neurally mediated)
 1. **vasovagal**: triggered by emotion, pain or stress. Often referred to as 'fainting'
 2. **situational**: cough, micturition, gastrointestinal
 3. **carotid sinus syncope**
- 2) Orthostatic syncope
 1. **primary autonomic failure**: Parkinson's disease, Lewy body dementia
 2. **secondary autonomic failure**: e.g. Diabetic neuropathy, amyloidosis, uraemia
 3. **drug-induced**: diuretics, alcohol, vasodilators
 4. **volume depletion**: haemorrhage, diarrhoea
- 3) Cardiac syncope:
 1. **arrhythmias**:
 - ⇒ bradycardias (sinus node dysfunction, AV conduction disorders) or
 - ⇒ tachycardias (supraventricular, ventricular)
 2. **structural**:
 - ⇒ valvular,
 - ⇒ myocardial infarction,
 - ⇒ hypertrophic obstructive cardiomyopathy
 3. others: **pulmonary embolism**

Reflex syncope is the most common cause in all age groups although orthostatic and cardiac causes become progressively more common in older patients.

Evaluation:

- 1) Cardiovascular examination
- 2) postural blood pressure readings: a symptomatic fall in
 - ⇒ **systolic BP > 20 mmHg** or
 - ⇒ **diastolic BP > 10 mmHg** or
 - ⇒ decrease in **systolic BP < 90 mmHg** is considered diagnostic
- 3) ECG
 - In the acute situation the ECG is the most useful test for classifying syncopal episodes into high risk and low risk categories.
 - High risk cases are those of abrupt onset occurring in patients older than 55 years who have a history of heart disease or abnormal ECG.
 - Low risk patients are those who have no underlying diseases and a normal ECG.
- 4) carotid sinus massage
- 5) tilt table test
- 6) 24 hour ECG

Pacemaker

Pacemaker Components

Pacemakers consist of:

1. Pulse generator

- Power source
- Battery
- Control circuitry
- Transmitter / Receiver
- Reed Switch (Magnet activated switch)

2. Lead(s)

- Single or multiple
- Unipolar or bipolar

Pacemaker Classification

- Pacemakers are classified by the nature of their pacing mode.
- The code is expressed as a series of up to five letters.

NBG Pacemaker Code (2002)

I	II	III	IV	V
Chamber(s) Paced	Chamber(s) Sensed	Mode(s) of Response	Rate Modulation	Multisite Pacing
O = None	O = None	O = None	O = None	O = None
A = Atrium	A = Atrium	T = Triggered	R = Rate modulation	A = Atrium
V = Ventricle	V = Ventricle	I = Inhibited		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)	D = Dual (T+I)		D = Dual (A+V)

Position I: Chambers Paced

- Refers to chambers paced.

Position II: Chambers Sensed

- Refers to the location where the pacemaker senses native cardiac electrical activity.

Position III: Response to Sensing

- Refers to pacemakers response to sensed native cardiac activity.
- T = Sensed activity results in triggering of paced activity
- I = Sensed activity results in inhibition of pacing activity

Position IV: Rate Modulation

- Indicates ability for rate modulation designed to altered heart appropriately to meet physiological needs e.g. physical activity.
- Sensors may measure and respond to variables including vibration, respiration, or acid-base status.

Position V: Multisite Pacing

- Allows indication of multiple stimulation sites within one anatomical area e.g. more than one pacing site within the atria or biatrial pacing

Common Pacing Modes

AAI

- Atrial pacing and sensing.
- If native atrial activity sensed then pacing is inhibited.
- If no native activity sensed for pre-determined time then atrial pacing initiated.
- Used in **sinus node dysfunction** with **intact AV** conduction.
- Also termed atrial demand mode.

VVI

- Ventricle pacing and sensing.
- Similar to AAI mode but involving ventricles instead of the atrium.
- Used in patients with chronic atrial impairment e.g. **atrial fibrillation** or **flutter**.
- Also in NYHA Class III symptoms and severe left ventricular systolic dysfunction.

DDD

- Capable of pacing and sensing both atrium and ventricles.
- Commonest pacing mode.
- Atrial pacing occurs if no native atrial activity for set time.
- Ventricular pacing occurs if no native ventricle activity for set time following atrial activity.
- Atrial channel function is suspend during a fixed periods following atrial and ventricular activity to prevent sensing ventricular activity or retrograde p waves as native atrial activity.

Magnet mode

- Applying a magnet to a pacemaker will initiate the magnet mode.
- This mode varies with pacemaker set-up and manufacturer.
- Usually initiates an asynchronous pacing mode – AOO, VOO, or DOO.
- Asynchronous modes deliver constant rate paced stimuli regardless of native rate of rhythm.
- In asynchronous ventricle pacing there is a risk of pacemaker-induced ventricular tachycardia.
- Note this differs from magnet application to an Implantable Cardioversion Defibrillator (ICD) which results in defibrillator deactivation.

Paced ECG – Electrocardiographic Features

The appearance of the ECG in a paced patient is dependent on the pacing mode used, placement of pacing leads, device pacing thresholds, and the presence of native electrical activity.

Features of the paced ECG are:

Pacing spikes

- Vertical spikes of short duration, usually 2 ms.
- May be difficult to see in all leads.
- Amplitude depends on position and type of lead.
- Bipolar leads result in a much smaller pacing spike than unipolar leads.
- Epicardially placed leads result in smaller pacing spikes than endocardially placed leads.

Atrial Pacing

- Pacing spike precedes the p wave.
- Morphology of p wave dependent of lead placement but may appear normal.

Ventricular Pacing

- Pacing spike precedes the QRS complex.
- Right ventricle pacing lead placement results in a QRS morphology similar to LBBB.
- Left epicardial pacing lead placement results in a QRS morphology similar to RBBB.
- ST segments and T waves should be discordant with the QRS complex i.e. the major terminal portion of the QRS complex is located on the opposite side of the baseline from the ST segment and T wave.

Dual Chamber Pacing

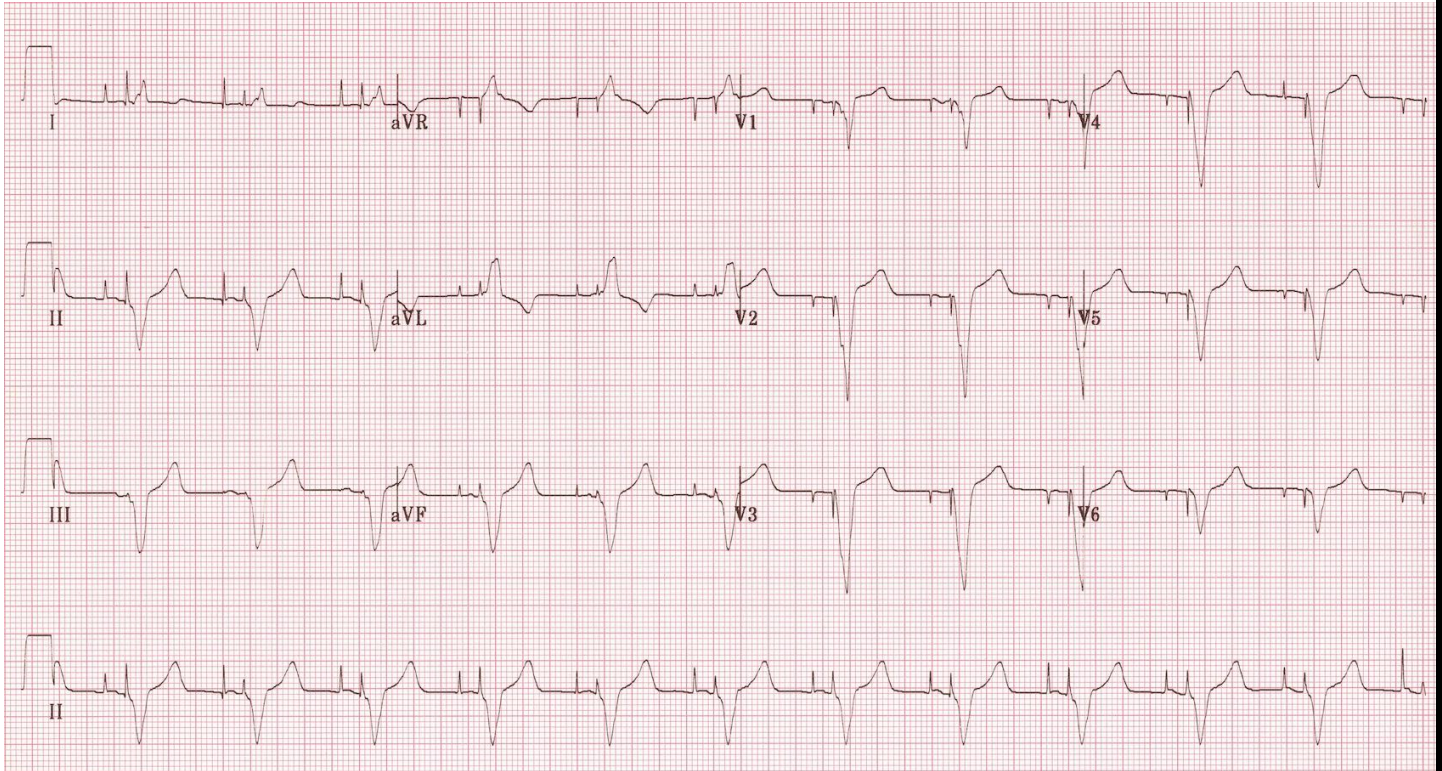
- Dependent on areas begin paced.
- May exhibit features of atrial pacing, ventricular pacing or both.
- Pacing spikes may precede only p wave, only QRS complex, or both.

The absence of paced complexes does not always mean pacemaker failure as it may reflect satisfactory native conduction.

ECG Examples

Dual Chamber Pacing

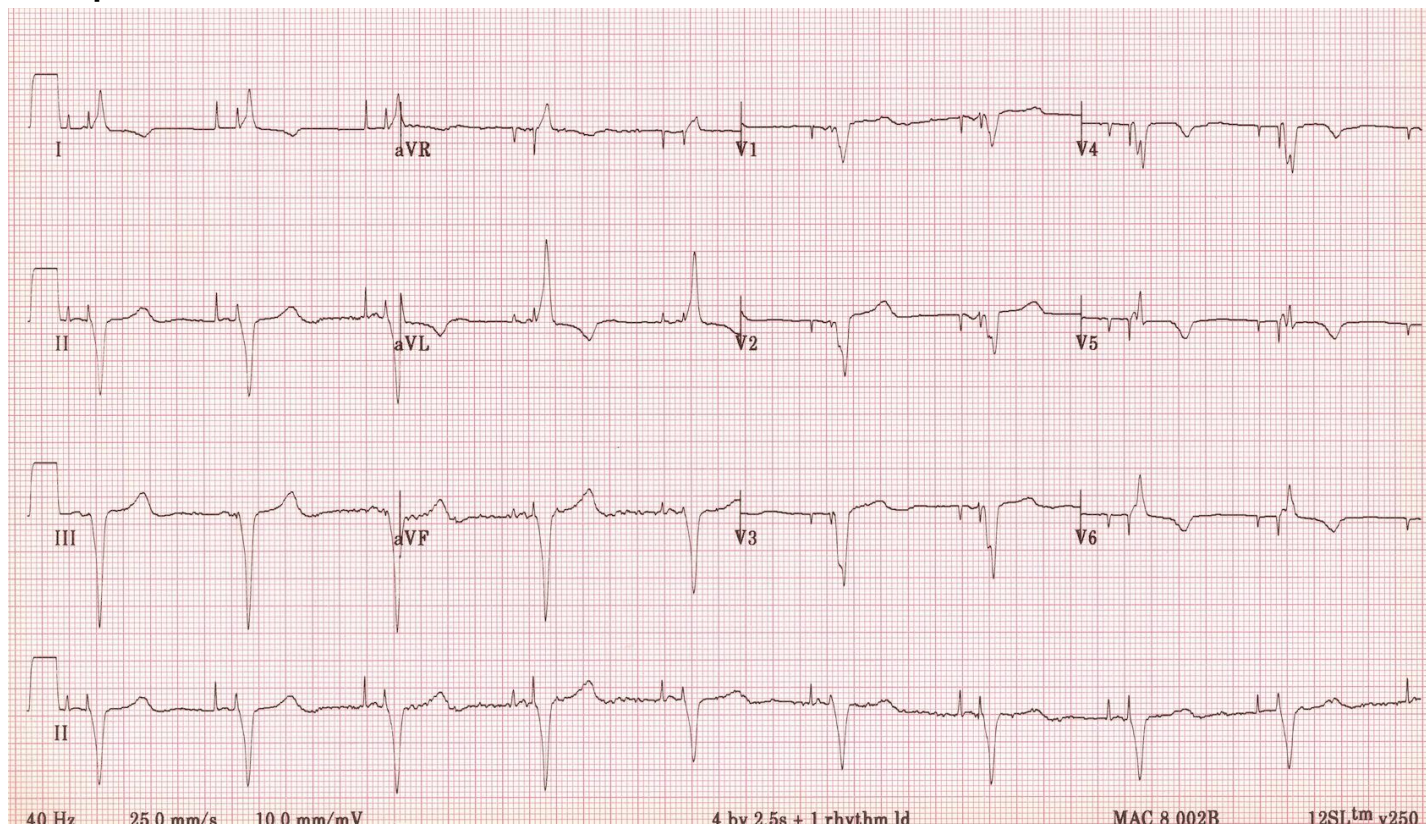
Example 1



A-V sequential pacing:

- Atrial and ventricular pacing spikes are visible before each QRS complex.
- There is 100% atrial capture — small P waves are seen following each atrial pacing spike.
- There is 100% ventricular capture — a QRS complex follows each ventricular pacing spike.
- QRS complexes are broad with a LBBB morphology, indicating the presence of a ventricular pacing electrode in the right ventricle.

Example 2



- **A-V sequential pacing — atrial and ventricular pacing spikes precede each QRS complex with 100% capture.**

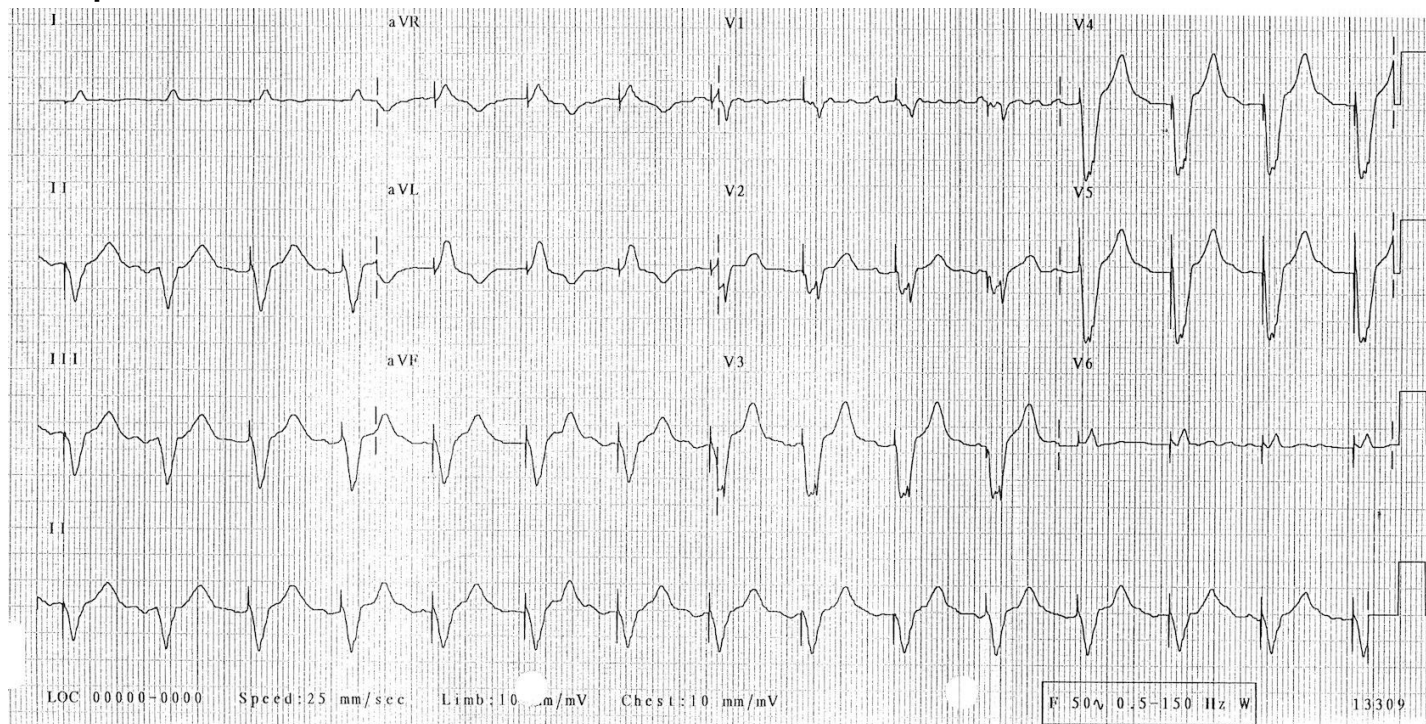
Example 3



- **Another example of A-V sequential pacing.**

Ventricular Pacing

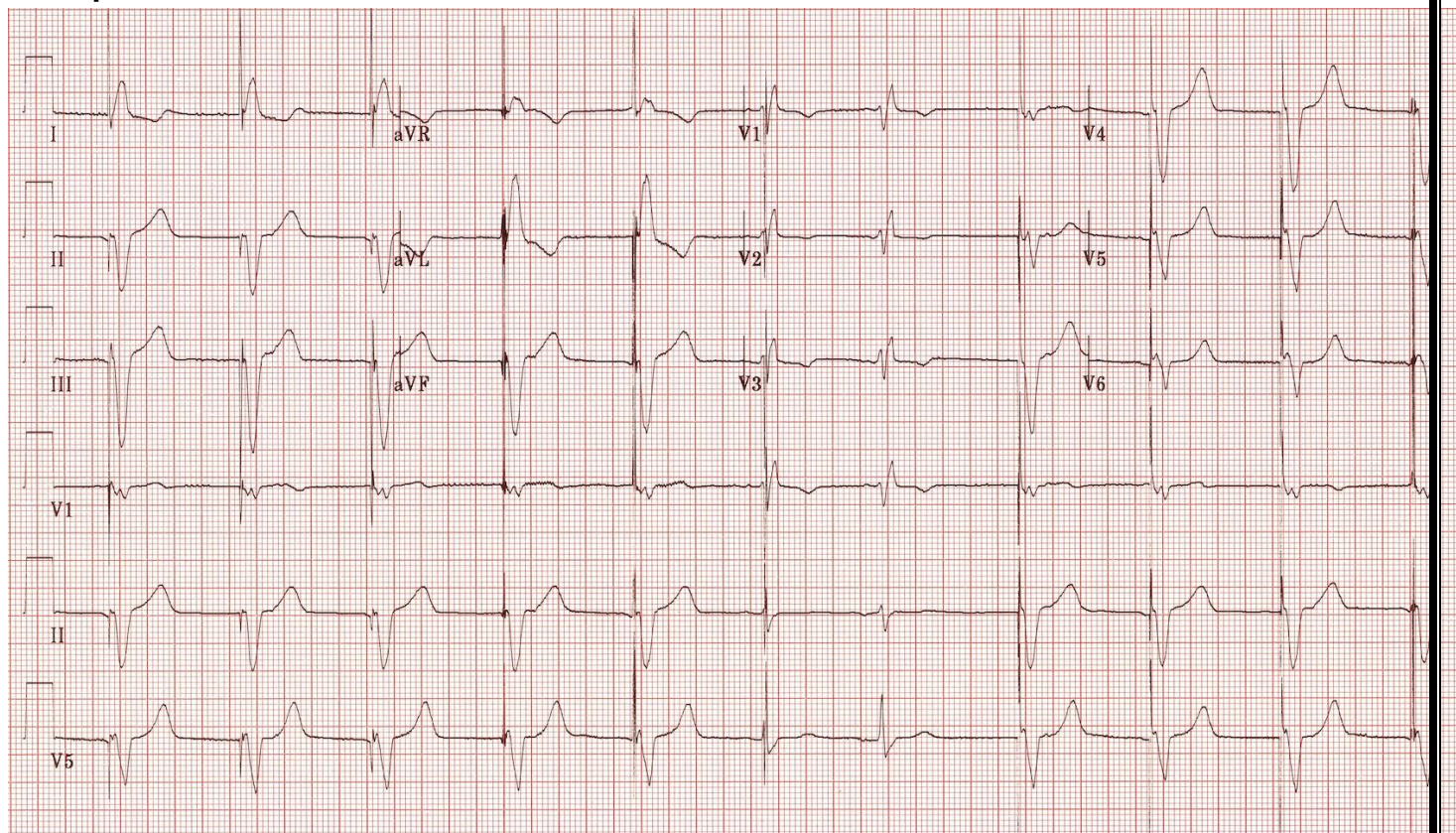
Example 4



Ventricular paced rhythm:

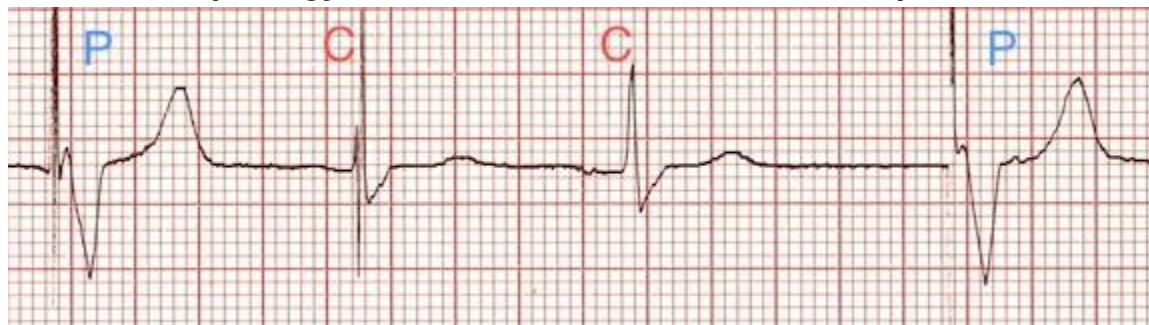
- Ventricular pacing spikes precede each QRS complex (except perhaps complex #2 — although the QRS morphology in this complex is identical to the rest of the ECG, suggesting that this beat is also paced)
- No atrial pacing spikes are seen.
- The underlying native rhythm is probably coarse atrial fibrillation — there are several possible P waves visible in V1 but otherwise the atrial activity is chaotic.

Example 5



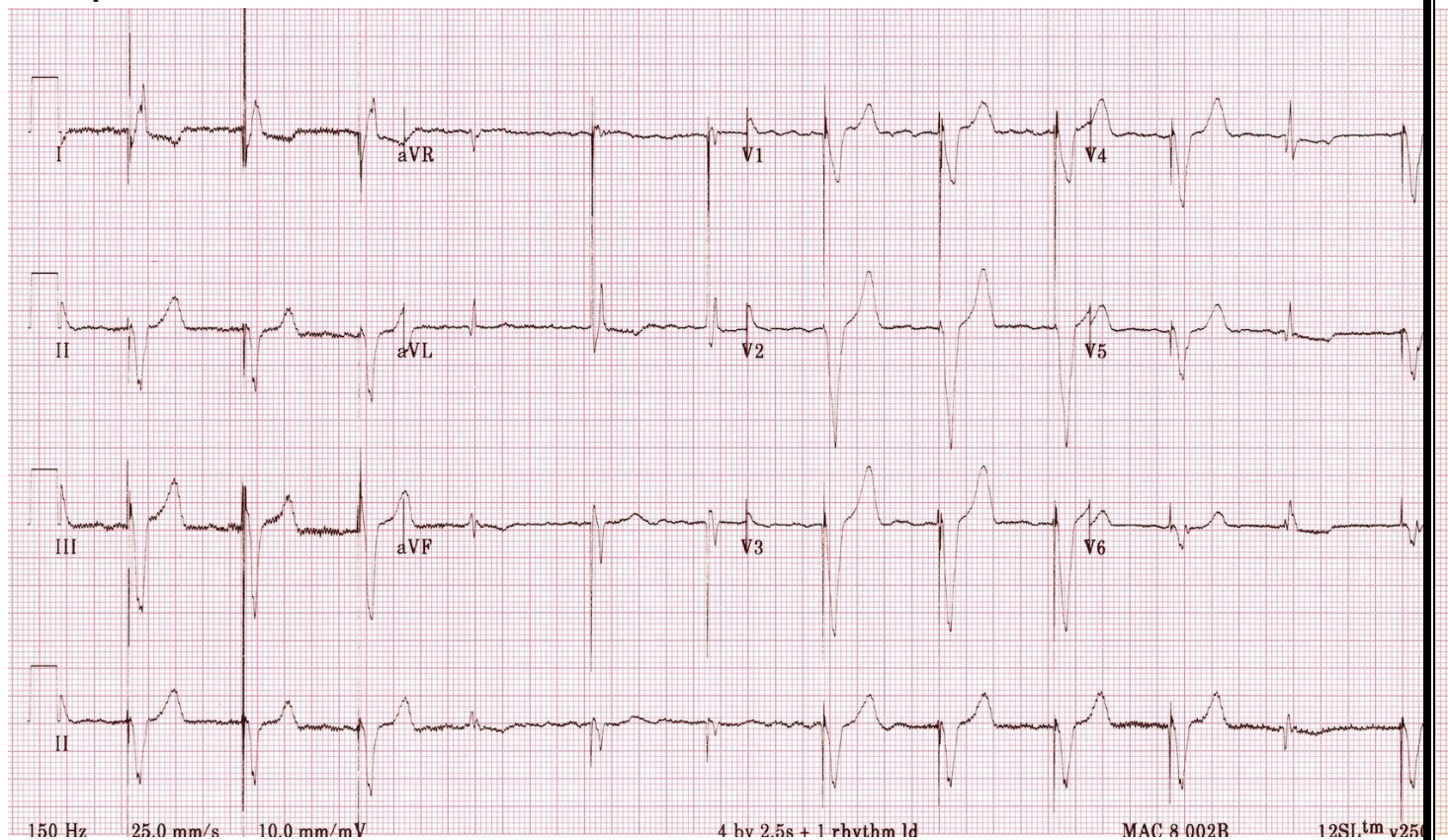
Ventricular paced rhythm:

- Ventricular pacing spikes precede most of the QRS complexes.
- The 6th and 7th beats are narrower, with a different morphology — these are non-paced (“capture”) beats, probably supraventricular in origin.
- There is a pacing spike superimposed on beat #6, but this does not appear to alter its morphology — i.e. no evidence of a fusion complex.



P = paced beat, C = capture beat

Example 6



Ventricular paced rhythm:

- Ventricular pacing spikes precede the QRS complexes, most of which exhibit LBBB morphology consistent with a RV pacing electrode.
- The 5th, 6th and 11th complexes are narrower with a different morphology — these are fusion beats produced when the ventricle is simultaneously activated by both paced and supraventricular (native) impulses. You can see how the pacing spike is shortened and the QRS duration narrowed by the co-incident native impulse.
- The 4th complex is probably a supraventricular capture beat, although there may still be some hybridisation with a pacing spike.

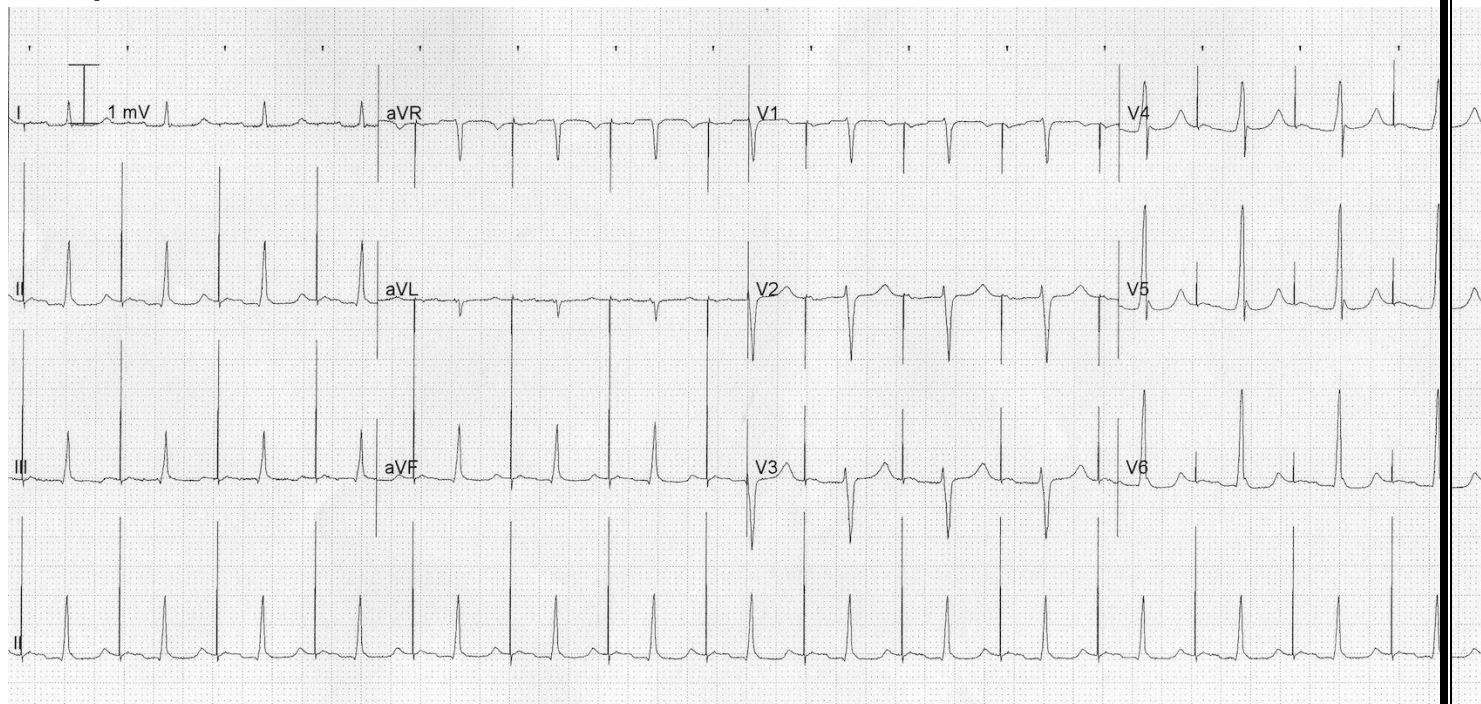


P = paced beat, F = fusion beat, C = capture beat

Atrial Pacing

Atrially paced patients often have evidence of 1st degree AV block or Wenckebach conduction on their paced ECG that is not apparent on their baseline tracing. This is because the sort of patients that require atrial pacing (e.g. post-op cardiac surgery) commonly have some degree of AV node dysfunction (e.g. due to age-related AV-nodal degeneration / their underlying cardiac condition / post-operative ischaemia / AV-nodal blocking drugs). When these patients are paced at a faster rate than their AV node can handle, the AV node becomes “fatigued” resulting in 1st degree AV block or Wenckebach phenomenon on the paced ECG. This abnormality is not clinically important provided that the patient’s cardiac output is not compromised.

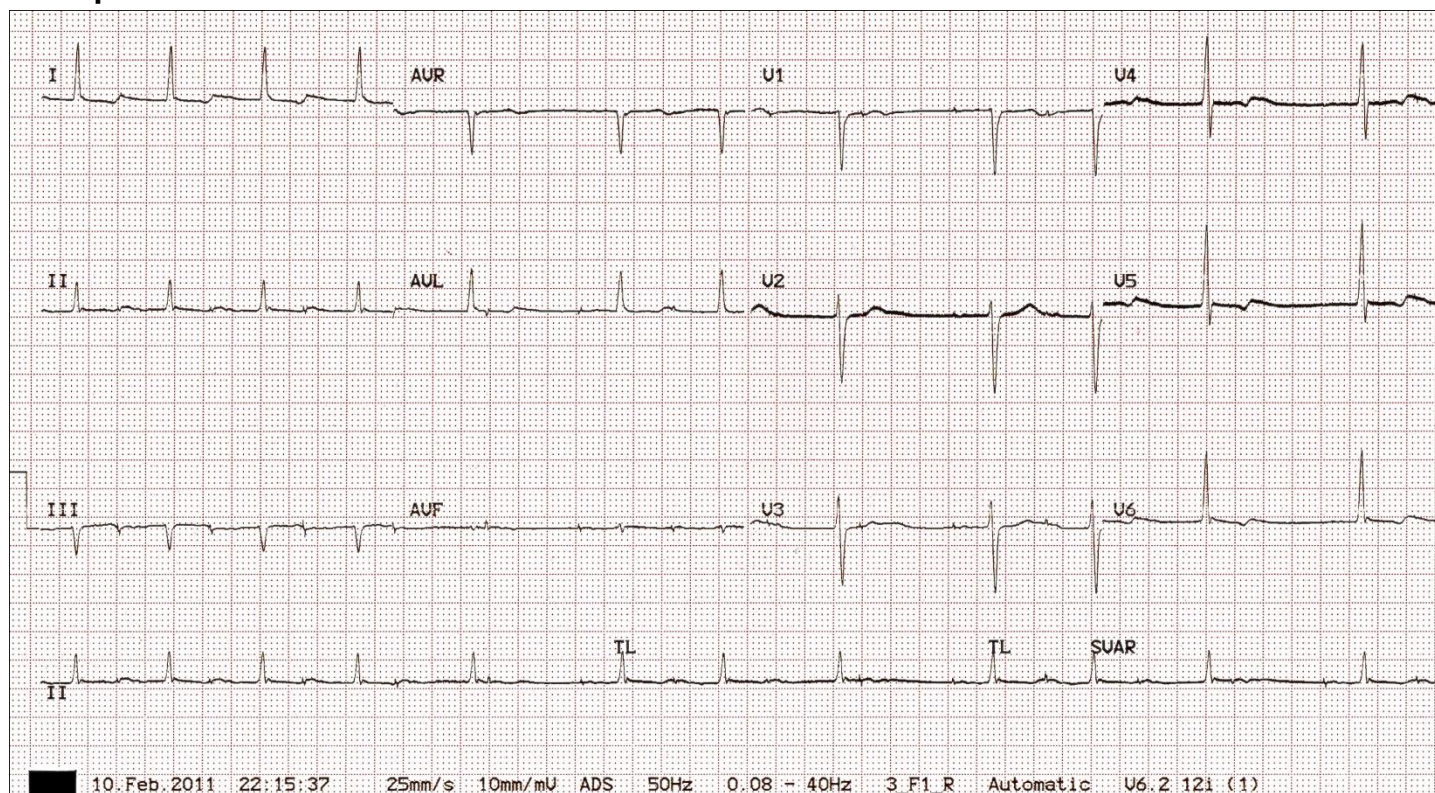
Example 7



Atrial paced rhythm with 1st degree AV block:

- There are regular pacing spikes at 90 bpm.
- Each pacing spike is followed by a P wave, indicating 100% atrial capture.
- P waves are conducted to the ventricles with a prolonged PR interval (280 ms).

Example8



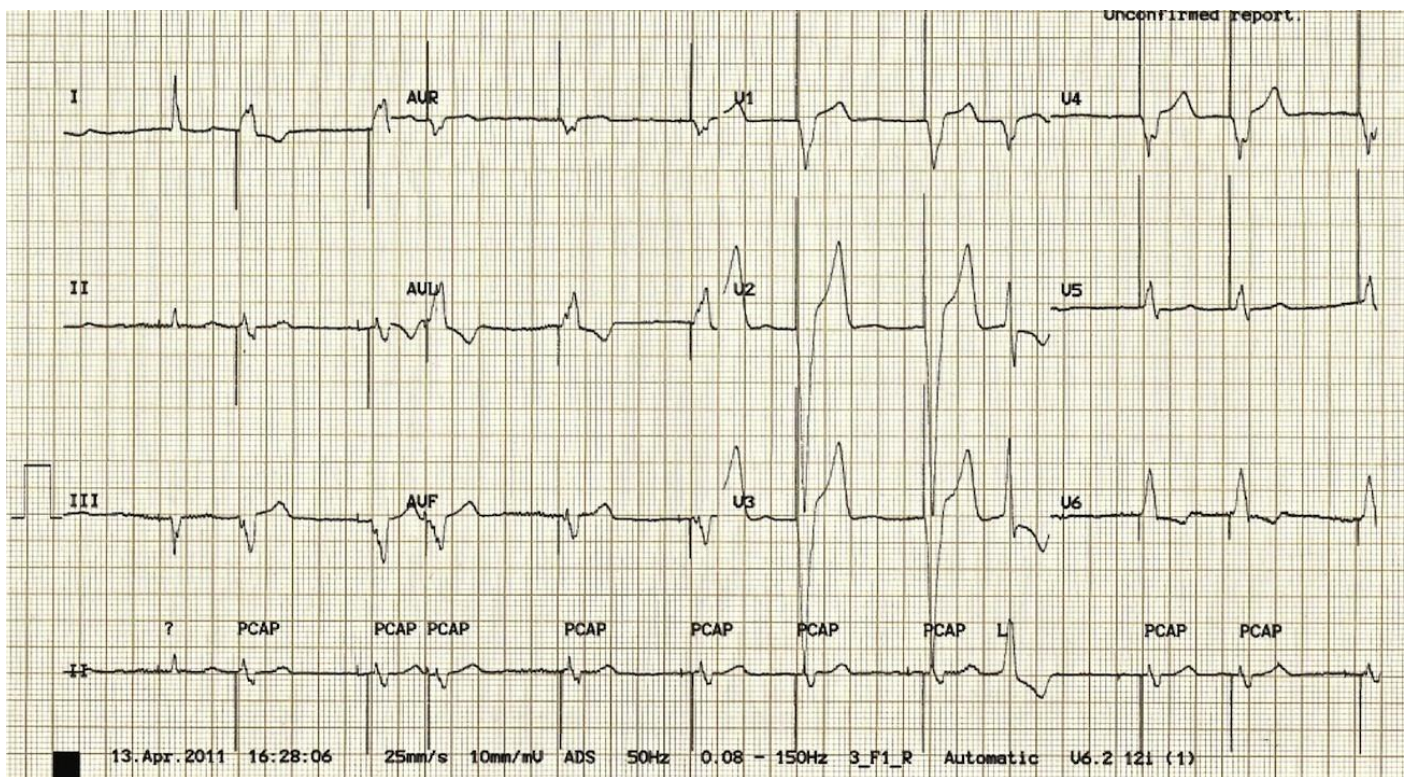
Atrial paced rhythm with Wenckebach conduction:

- There are regular atrial pacing spikes at 90 bpm; each one is followed by a small P wave indicating 100% atrial capture.
- However, not every P wave results in a QRS complex — the PR interval progressively lengthens, culminating in failure of AV conduction (“dropped QRS complexes”).
- There is a co-existent 2nd degree AV block with Mobitz I conduction (Wenckebach phenomenon).
- The Mobitz I is due to the patient being paced at a rate faster than his AV node can handle — at his own intrinsic rate of 50-60 bpm he had only a 1st degree AV block.

To find out the story behind this ECG, check out [ECG Exigency 011](#).

Pacemaker Puzzler

Can you work out what is going on in this ECG?



الصاعقة (البرق)

The figure shows a 'ferning' or 'arborescent' rash pathognomonic of a lightning strike, also known as Lichtenberg figures.

"The pathology of lightning, or keraunopathy, is known only to a few specialists."¹

Reference:

1. NASA Science. Human Voltage: What happens when people and lightning converge.